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ANNALS OF INTERNAL MEDICINE

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DEPARTMENT OF REVIEWS

The Journal will make an especial feature of the review of monographs and books bearing upon the field of Internal Medicine. Authors and publishers wishing to subject such material for the purposes of review should send it to the editor. While obviously impossible to make extended reviews of all material, an acknowledgment of all matter sent will be made in the department of reviews.

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Insulin and Carbohydrate Tolerance*

By Elliott P. Joslin, M.D., *Boston*

In the same serious spirit with which Brace[1] approached the problem of the effect of insulin upon the carbohydrate tolerance of diabetics, I have searched my own records and have reached a contrary and more optimistic conclusion and, I trust, explained his less favorable results. If he should reduce the total caloric intake of his patients to levels such as are accepted as normal for healthy individuals, thus involving a reduction of fat in the diet he prescribed, I believe he would be able to increase the carbohydrate in the course of a year with negligible or little increase in insulin, and thus demonstrate a gain in carbohydrate tolerance of diabetics under insulin medication.

As Brace has done and as I am now doing, it would be helpful to an understanding of the present status of diabetic treatment if other clinicians, interested in diabetes, would also publish comparable series of five diabetics who have exceptional records over an interval of at least four years.

"A failure to obtain evidence of improvement in the tolerance for glucose in five patients, who had received weighed, high fat, low protein and low carbohydrate diets and daily insulin over periods varying from 32 to 45 months, has given us no ground for believing that insulin is capable of effecting a cure or a partial cure of human diabetes mellitus. * * * Each of these patients has lost tolerance during the period of treatment with insulin."[1]

Frankly I cannot agree with the above quotation from the conclusions of Brace's article. The protocols of his patients show that they were not receiving "low protein and low carbohydrate diets" in the Newburg and Marsh[2] sense (See Table 1) and the caloric values of the diet were excessive by any standard. Furthermore, support for my disagreement is afforded by the remarkably favorable course for two years of his Case V while on a constant diet of carbohydrate 64 grams, protein 66 grams, and fat 255 grams, because during this interval his daily insulin was re-

Table 1—The Original Newburgh Marsh Diets

Diets	C. Grams	P. Grams	F. Grams	Calories
1	15	20	86	914
2	20	28	129	1353
3	29	34	163	1719
4	37	54	210	2254

*From the New England Deaconess Hospital, Boston.

[1]Brace: Annals of Internal Medicine, 1927, 1, 203.

[2]Newburg and Marsh: Arch. Int. Med., 1920, *26*, 647; also ibid, 1923, *31*, 455.

TABLE 2—A REDUCTION OF 16 UNITS OF INSULIN IN 2 YEARS DURING TREATMENT OF A DIABETIC UPON A CONSTANT DIET

Case V, F. J., Cited from Brace.[1] Italics inserted by Author.

Date	Diet P. Gms.	F. Gms.	C. Gms.	Total Glucose Gms.	Insulin Units	Urine Glucose
July 1919	53	210	37	90	0	Neg.
Mar. 1923	53	210	37	90	0	Neg.
Mar. 1923	70	290	75	144.6	26(13-13)	Neg.
Apr. 1924	70	290	75	144.6	46(16-15-15)	Neg.
Aug. 1924	70	290	75	144.6	41(21-10-10)	Neg
Dec. 1924	70	290	75	144.6	40(20-8-12)	Neg.
Jan. 1925	*66*	*255*	*64*	*127.7*	*42(20-9-13)*	*Neg.*
June 1925	*66*	*255*	*64*	*127.7*	*34(13-8-13)*	*Neg.*
Aug. 1925	*66*	*255*	*64*	*127.7*	*27(13-5-9)*	*Neg.*
Jan. 1927	*66*	*255*	*64*	*127.7*	*26(12-5-9)*	*Neg.*

duced from 42 units to 26 units! See Table 2.

I am not at all surprised that this same patient, Case V, irrespective of infections, increased his insulin in an earlier period between March 1923 and January 1925 At the former date upon recovery from an infection and a semi-comatose state this man, 30 years old, weight 45 kilograms, was given *50*(!) calories per kilogram body weight (C. 37, P. 53, F. 210, Cal. 2250) and before the end of the same month *71*(!) calories per kilogram (C. 75, P. 70, F. 290, Cal. 3190) and this diet was maintained until January 1925. Thereupon carbohydrate, protein and fat were all reduced and the striking gain in tolerance ensued. In about four years the weight rose from 45 kilograms to 55 kilograms or 21 per cent.

At no time was this patient or in fact any of the five cases cited by Brace on a low protein diet unless in the brief unpublished test periods. See Table 2. For their final diets the protein was approximately 1 gram per kilogram body weight. The caloric values of the diets were also high and, as last recorded, were 42.9, 45.6, 40.5, 39.1, and 51.5 calories respectively per kilogram body weight. The success of the original Newburg-Marsh diets, which are shown in Table 1, can be ascribed largely to their low caloric values, quite as much as to their low carbohydrate and low protein values, but in the diets of the patients reported by Brace low protein, and indeed low carbohydrate in four of the five cases, have been discarded and the total calories have been raised considerably above the needs of healthy adults. Diabetes comes on with overfeeding rather than with underfeeding and it is against a fundamental law of diabetes to expect gains in tolerance when a patient is overfed. Under these circumstances I consider it remarkable that the patients of Brace made so good a showing, and instructive that Case V should have improved so much as to have been able to reduce his insulin 16 units a day in the course of 2 years.

TABLE 3—LAST RECORDED DIETS OF THE DIABETIC SERIES OF BRACE

Case No.		Age Years	Weight Kilog.	Diet in Grams C.	P.	F.	Total Glucose	Calories per Kilog.	Total Units	Insulin Carb. per unit Gms.	Total Glucose per unit Gms.	Cal. per kilog. per unit
I	L.C.	21	54.5	35	55	220	88.9	42.9	56	0.6	1.6	0.8
II	R.B.	32	60.0	60	60	280	122.8	45.6	85	0.7	1.4	0.5
III	R.K.	20	61.0	45	55	230	99.9	40.5	30	1.5	3.3	1.4
IV	D.A.H.	28	63.0	60	60	220	116.8	39.1	52	1.2	2.3	0.8
V	F.J.	34	54.5	64	66	255	127.7	51.5	26	2.5	4.9	2.0

The unit values for insulin in terms of carbohydrate, total glucose, and calories per kilogram are shown for Brace's cases in Table 3. Save for Case V these values seem strikingly low. In only one case was the unit value for carbohydrate above 1.5 grams, and in but 2 instances did the total glucose value exceed 2.3 grams. Two patients obtained but 0.6 and 0.7 gram carbohydrate per unit of insulin and 1.6 and 1.4 grams total glucose per unit. Despite the high caloric diets which these patients received the calories per kilogram body weight per unit insulin were meagre, amounting to 0.8, 0.5, 1.4, 0.8, and 2.0 calories respectively.

These five patients of Brace certainly obtained small returns upon their investments in insulin and this leads me now to present the other side of the question.

It is difficult for me even in the abstract to accept continued loss of tolerance or failure to gain tolerance for carbohydrate in diabetes because of the following considerations.

(1) If diabetics grow worse with time, what is the explanation for the absence of "complete" diabetes, which would seem to be the goal that all diabetics must reach, with that premise, among my 161 patients of 20 or more years' duration? If diabetics "distinguished * * * for their loyalty, honesty, intelligence" (Brace) lose tolerance, what must be the fate of the ordinary diabetic? At what rate is tolerance lost by the exemplary diabetic and by how much must one expect the rate to be accelerated in the careless?

Even careless diabetics sometimes do extraordinarily well, although I hardly dare acknowledge it. These patients in general are not susceptible to careful investigation and the only proof of the proposition is in length of life, appearance, and activity. I will mention 4 such patients by their Case numbers, 428, 1254, 1609, 1895, all of whom are described elsewhere.[3] The diabetes of Case No. 1609 began in 1917 when he was 10.7 years old. It was severe in type, his weight fell from 90 to 71½ pounds during the first two years. He broke his diet continuously and it seemed a miracle that he escaped death from coma. Even with insulin he was seldom

[3] JOSLIN: Treatment of Diabetes Mellitus. 4th edition, Lea & Febiger, Philadelphia, 1928. Consult Case Index.

sugar-free, but 10 years later in February 1927, he appeared in my office as an attractive, healthy looking young man of twenty years, 5 feet 7¾ inches tall, and weighing 135 pounds. His urine contained no sugar, the blood sugar eight hours after a meal was 0.21 per cent, and he was taking 10 units of insulin in the morning, 10 at noon, and 16 at night. He was working hard, carrying on his own small farm and is alive January, 1929.

(2) It is my general impression after contact with diabetics for 30 years that if they follow reasonable rules of treatment the longer they live the milder their disease becomes. My cases of long duration die of the complications of diabetes rather than of coma, and the patients who develop coma always give a history of gross indiscretions. Neither Von Noorden with his 20,000 diabetics, nor I with 7,000 believe we have encountered a complete diabetic and the few cases of insulin refractory diabetes in the literature tend to confirm the truth of our opinions. I have seen one case, Case No. 6247, of hitherto unexplained refractory diabetes,[4] but even that case is now explained as one of hemochromatosis as the result of an autopsy.

Diabetic mortality among individuals under 20 years of age in Massachusetts has almost reached the vanishing point,[5] and implies that the few who die are dying needlessly, and yet this is the group of diabetics which is supposedly severe and should become complete diabetics if their tolerance were fading away.

(3) My diabetic children daily challenge pessimism by their good looks, their vigor of mind and body, and their normal sexual development. The return of menstruation in Case No. 2617 at the age of 20 years after 6.3 years of diabetes occurred in June 1928, 6.5 years after it last took place. This has encouraged me very much, because I think she was one of the few exceptions I had among this type of patients. If tolerance was failing, would one expect catamenia to begin? As pulmonary tuberculosis advances, does menstruation come back? The failure of diabetes to be fatal in more than six instances among 303 of my children during the 22 months ending July 1, 1928 does not speak in favor of loss of tolerance.[6]

(4) The above considerations are largely general in nature. Since I have claimed so often that the tolerance in diabetes can improve, it is manifestly my duty to furnish examples. Like Brace I will cite five cases. I frankly admit that I have not like Brace omitted the insulin which these patients are taking or reverted to their original test diets in order to test their tolerance and to compare it with that at the first observation, and moreover I am unwilling to do so.

Brace describes his method as follows:[1] "The glucose tolerance of each individual is always determined by beginning with bed rest and a daily diet consisting of protein 20 grams, fat 86 grams, and carbohydrate 15 grams, without insulin. The total glucose con-

[4] JOSLIN: ibid, p. 634.
[5] JOSLIN: ibid, p. 121.
[6] JOSLIN AND WHITE: Jour. Am. Med. Assn., 1929, 92, 143.

tent of this diet is 30 grams. After an aglycosuric period of three to four days, each foodstuff is proportionately increased so that the next diet has a total glucose content of 50 grams and consists of protein 28 grams, fat 129 grams, and carbohydrate 20 grams. Similarly, after aglycosuria periods of the same time length, the diet is raised proportionately until its total glucose content is 70 grams, consisting of protein 34 grams, fat 163 grams, carbohydrate 29 grams. The fourth level affords 90 grams of total glucose, consisting of protein 54 grams, fat 210 grams, and carbohydrate 37 grams. To the latter diet carbohydrate is added in ten gram measures every third or fourth day, until there is a glycosuria."

I realize perfectly well that some will say my standards for the determination of the present tolerance are not as scientific as the careful regime to which Brace subjected his patients. But is his test as scientific as it seems? His patients underwent comparable tests at beginning and end of the periods of observation, it is true, but note (a) the preliminary diets for the tests were undernutrition diets whereas the endogenous metabolisms of the patients at the beginning and end of the average three years' interval were dissimilar and indeed not comparable, because of differences in body weights at the two periods, which were due to deposits of fat rather than to growth; (b) upon both occasions the stimulating effect of exercise upon carbohydrate tolerance was lost, because the patients were placed in bed, and the tests were therefore artificial; (c) the sudden restriction in diets which the tests involved itself tends to lower or at least conceal tolerance for carbohydrate as proved by Odin[7] even for normals. This also exerted more of an influence in the second testing because the return to the diet of protein 20 grams, fat 86 grams, and carbohydrate 15 grams represented a relatively greater lowering of caloric intake than at the first test when the weights of the patients and their needs were less; (d) no adequate opportunity was given the patients to disclose their capacity for a growing tolerance. To develop a tolerance for carbohydrate it is essential that slow, gradual, and very small additions to the carbohydrate intake must be undertaken rather than sudden additions even of 10 grams. If the carbohydrate in the diet is 37 grams an increase of 10 grams amounts to an addition of 27 per cent! If my patients could gain one gram of tolerance for carbohydrate a month what an advance that would represent compared with pre-insulin days!

A comparison of the ages of onset of the diabetes, the duration of the disease, the diets, the carbohydrate, glucose and calorie-kilogram equivalent of an unit of insulin is given in Tables 2, 4, and 5 for the five cases selected by Brace and the 5 from my series.

The ages of onset of my cases average 15 years, 8 years less than the cases of Brace, and the durations of the disease in my series average 12 years in contrast to 4 years for those

[7] Odin: Acta Medica Scand: 1927, Supp. 18, 388.

TABLE 4—COMPARISON OF THE TWO GROUPS OF DIABETICS OF BRACE AND JOSLIN

Brace	Onset		Duration of Disease to Final Observation	
	Date	Age	Date	Duration
I (L.C.)	1922 June	18	1926 Jan.	3.6
II (R.B.)	1922 Feb.	28	1926 March	4.1
III (R.K.)	1924 Jan.	18	1926 March	2.2
IV (D.A.H.)	1923 Aug.	25	1926 April	2.7
V (F.J.)	1919 Jan.	26	1927 Jan.	8.0
	Average (years)	23	Average (years)	4.1

Joslin	Onset		Duration of Disease to Final Observation	
	Date	Age	Date	Duration
359	1910 Feb.	33	1929 Jan.	18.9
632	1912 Aug.	30	1929 Jan.	16.3
1616	1919 Aug.	5.5	1929 Jan.	9.4
2084	1919 June	3.2	1928 Dec.	9.5
2560	1921 June	5	1928 Aug.	7.2
	Average (years)	15.3	Average (years)	12.3

TABLE 5—LAST RECORDED DIETS OF THE DIABETIC SERIES OF JOSLIN

Case No.	Age Years	Weight Kilog.	Diet in Grams C.	P.	F.	Calories per Kilog.	Total Glucose	Total Units	Insulin Carb. per unit Gms.	Total Glucose per unit Gms.	Cal. per kilog. per unit
359	52	56.0	236	116	95	40.	313.3	15	15.7	20.9	2.7
632	46	59.0	166	69	119	34.	218	26	6.4	8.4	1.3
1616	14	32.3	128	84	132	63.	190	28	4.6	6.8	2.3
2084	13	28.4	110	85	90	56.	168	27	4.1	6.3	2.1
2560	12	33.9	125	65	125	56.	175	22	5.7	8.0	2.5

of Brace. The youthfulness of my patients attests to some extent their severity, but to a less degree than is ordinarily supposed. It may help to explain their gain in tolerance, because a young pancreas may be more capable of regeneration than an old pancreas. Obviously the younger age of the patients accounts for the relatively high protein and caloric values per kilogram body weight. The high protein value of Case No. 359 is in fact explained by his unusual activity in the post-office at the Christmas season.

AUTHOR'S CASES

Case No. 359 developed diabetes at the age of 33 years in February 1910 and came under my observation in August of that year, his naked weight having fallen from 71 to 59 kilograms. He gave a history of "typhoid-malaria" in the Spanish war in 1898 and of jaundice in 1904 preceding the removal of a cystic left kidney by M. H. Richardson in February, 1905. During a part of these 19 years he has been a patient of Francis W. Palfrey whose records sup-

plement my own and to whom I am greatly indebted. The percentage of glycosuria about one month after onset and 8 days after the diagnosis of diabetes was 3.0 per cent on March 12, 1910 according to Dr. Palfrey's records. The first percentage reported to the patient was 6 per cent. He worked in the Boston Post Office but was transferred to another office and moved his home near to it. Upon June 28, 1927, his wife wrote my associate Howard Root—

"Dear Sir:

"I have such good news today I must tell you the credit belongs to you. Mr. B. received his report from U. S. Post Office department for year's work 99 per cent. They never mark 100 and very rarely 99. This includes moral of whole office, neatness of reports, daily financial condition and good will of the people in the district, and this report was made by their Washington inspectors who are trained in the business of looking for trouble. Oh! Doctor I love my scales . . . . ."

The husband and wife ate 2 bushels of carrots and 500 pounds of squash in the winter of 1919, but glycosuria appeared when the squash ripened and when canned vegetables were used instead of fresh vegetables. Washed vegetables were freely em-

TABLE 6

Case No. 359. Male. Onset at 33 years in Feb. 1910
Weight Aug. 1910, 59 kg. Weight Jan. 1929, 56 kg. Height 171 cm.

Date	Urine Per Cent	Blood Sugar Per Cent	C.	P.	F.	Total Glucose	Naked Weight Kg.	Insulin Units
Mar. 12, 1910	3.0	.....	...	...	...	...		0
Aug. 15, 1910	trace	.....	...	...	...	...		0
May 29, 1912	0	.....	...	...	...	...		0
May, 1915	0	.....	...	...	...	...		0
Jan. 2, 1920	0	.....	...	...	...	111	54.5	0
Feb. 8, 1923	0	0.20 3 hrs. p. c.	22	133	104	109	52.7	0
Mar. 7, 1923	0	.....	36	90	147	103	...	0
Nov. 7, 1923	0.4[1]	0.25 fasting	64	42	82	91[2]	44.5	
Nov. 13, 1923	0	0.22 "	78	49	121	118	43.2	8-0-3
Nov. 29, 1923	0	0.17 "	70	49	145	113	50.0	8-0-7
Dec. 13, 1923	0	0.17 "	70	49	145	113		8-0-7
Dec. 4, 1924	0	0.13 "	78	45	168	121	52.7	5-0-5
Apr. 9, 1925	0	0.21 "	98	55	167	147	50.9	5-0-4
June 18, 1925	0	0.09 "	88	55	167	137	51.6	7-0-5
Oct. 29, 1925	0	0.07 "	90	55	167	139	51.6	7-0-5
June 3, 1926	0	0.20 "	94	75	178	156	50.9	5-0-3
Feb. 3, 1927	0	0.05 3½ hrs. p. b.[3]	109	82	183	175		10-0-10
Feb. 24, 1927	0	0.06 5 hrs. p. b.	109	82	173	174		10-0-0
Aug. 30, 1927	0	0.18	109	82	173	174		10-0-5
Nov. 30, 1927	0	0.06 1½ hrs. p. b.	109	82	173	174		10-0-5
Mar. 7, 1928	0	0.08 1 hr. p. b.	126	82	149	189		10-0-5
May 17, 1928	0	0.08 2½ hrs. p. b.	151	91	118	216		10-0-5
June 14, 1928	0	0.24 1 hr. p. b.	170	95	115	237		10-0-5
Oct. 18, 1928	0	0.05 2 hrs. p. b.	185	91	118	250		10-0-5
Jan. 10, 1929	0	0.06 3 hrs. p. b.	236	116	95	313	55.9	10-0-5

[1]6 grams in 24 hours.
[2]Calculated after deduction of urine sugar.
[3]p. b. signifies after breakfast.

ployed. In November 1923 he said he had never missed a day's work on account of diabetes in the 13 years he had had it.

Despite his one kidney for 24 years his blood pressure is 110/20 and the urine contains but a very slight trace of albumin and the non-protein nitrogen in the blood is normal. Wassermann negative. No gall stones by symptoms or roentgen ray. Diabetic heredity absent.

It would appear that with the help of insulin, diet, and time, his tolerance for carbohydrate had risen from 22 grams to 236 grams.

My second patient, Case No. 632, is the one with the best data and if one does not concede he has gained tolerance from an examination of Table 7 there is no need of reading the balance of this paper. Details about his course have been published for years,[8] and in January 1929 he is in better condition than ever since onset. He had difficulty in becoming sugar free on carbohydrate 15 grams in 1913 at the age of 31, one year after the onset of his diabetes. Gradually the carbohydrate rose to 29 grams, but in 1922 it had fallen to carbohydrate 15 grams, protein 69 grams, fat 103 grams. With 26 units of insulin he was sugar free on January 2, 1929, throughout the entire day upon carbohydrate 166 grams, protein 69 grams, and fat 119 grams. The calories in the diet are 34 per kilogram body weight. The carbohydrate per unit insulin is 6.4 grams. The total glucose per unit 8.3 grams, and the calories per kilogram per unit are 1.3. In the summer of 1928 he was selected to play in an international golf match and won distinction.

Wassermann negative. Diabetic heredity absent.

Upon July 10-12, 1928 the fasting blood sugar was 0.14 per cent, the blood sugar at 11 A.M. 0.08 per cent and at one hour after lunch 0.16 per cent; the cholesterol was 229 mg. and the fatty acids 362 mg. per 100 c.c. On these days passed in a diabetic home the carbohydrate was 116 grams, slightly lower than usual during the summer because he was inactive, the insulin 24 units. As stated above his tolerance has continued to rise and with 26 units of insulin he tolerated on December 30, 1928, 166 grams carbohydrate instead of 15 grams as on November 30, 1922.

[8]JOSLIN: Treatment of Diabetes Mellitus, Loc. Cit. See Case Indices 3rd and 4th Editions, 1923, 1928.

TABLE 7

Case No. 632. Male, Onset at 30 years in August, 1912
Wgt. Apr. 1916, 57 kilog. Wgt. Nov. 30, 1922, 50 kilog. December 1928, 59 kilog.
Height 179 c.m.

Date	Urine Glucose	Diet in Grams Carb.	Pro.	Fat	Total Glucose Grams	Insulin Units
1916, April 12	0	26	84	130	88	0
1918, June 24	0	22	74	100	75	0
1921, July 28	0	18	67	87	66	0
1922, Nov. 30	trace	15	69	95	65	0
1922, Dec. 5	0	41	72	122	95	4 (2-0-2)
1923, May 23	0	43	78	150	103	10 (6-0-4)
1924, May 3	0	38	77	148	98	10 (10-0-0)
1925, May 10	0	46	75	134	103	18 (18-0-0)
1926, May 5	0	70	77	135	128	20 (14-0-6)
1927, June 3	0	122	71	116	175	20 (20-0-0)
1928, June 14	0	135	75	116	190	24 (18-0-6)
1929, Jan. 2	0	166	69	119	218	26 (16-0-10)

Case No. 1616, Freddie G., "the severest case in a child in the series treated with insulin,"[9] and repeatedly cited in former publications developed diabetes in August 1919 at 5.5 years of age and his state became so pathetic before the discovery of insulin that his parents told me to "do anything you want with Freddie, you can't make him any worse." His weight in July 1919 was 19.6 kg., but when he came under my observation two months later it was 16.1 kg. and his height 109 c.m. Wassermann negative. Heredity absent. His course is shown in Table 8. At present, January 11, 1929, he is a happy boy, weight 32.3 kg., height 132 cm., and evidently still growing. Throughout the winter he does not wear a hat. He stands well at school and plays the banjo and mandolin very well. With the help of 28 units of insulin his tolerance for carbohydrate has risen from 55 grams in 1920 and probably much less than 36 grams in December 1922 to 128 grams in 1929.

Case No. 2084, Buddy H., a pale listless child, taking soda and whiskey, came to me December 1, 1920 with multiple abscesses, and 3.0 per cent glycosuria which required 72 days of hospital treatment for me to banish. Diabetes began 18 months before when he was 3 years and 2 months old. He has constantly been under the care of excellent trained nurses, whom he has seen one by one successfully married. His weight in December was 11 kg., height 100 cm. The weight is now, December 31, 1928, 28.4 kg., and height 127 cm. Wassermann negative. Heredity absent.

In the 12 and 14 days respectively the soda and whiskey were eliminated and the minus carbohydrate balance began to be permanently positive on the sixteenth day of treatment. The urinary nitrogen in this period fell from 10.8 and 11.5 grams a day to 4.5 grams. With the help of 27 units of insulin his gain in tolerance for carbohydrate has been from 27 grams in March 1923 to 110 grams in December 1928.

[9] JOSLIN: Jour. Metab. Research, 1923, 2, 651; also Treatment Diabetes Mellitus, 4th Ed. p. 825.

TABLE 8

Freddie G., Case No. 1616, my severest child diabetic in November 1922. Age at onset in Aug. 1919, 5.5 years, Weight 16.1 kilog., Height 109 cm.; Weight March 17, 1922, 11 kilog.; Weight Jan. 1929 32.3 kilog., Height 132 cm.

Date	Urine Sugar	Diet C. Gms.	Diet P. Gms.	Diet F. Gms.	Total Glucose	Insulin Units
1919, Sept. 10	0	48	32	24	70	0
1920, March 20	0	30[1]	61	61	71	0
1922, Nov. 3	4.1%	...	...	...	...	..
1922, Nov. 6	19 grams	50	25	35	69	2
1922, Nov. 13	6 grams	41	37	50	68	12
1922, Dec. 4	0	36	37	68	65	12
1923, March 15	0	36	37	68	65	10
1924, Aug. 29	0	41	37	68	70	11.5
1925, May 2	0	44[1]	?50	?78	81	14
1926, May 23	0	...	...	...	...	22
1927, Feb. 7	0	101	73	118	156	23.5
1927, Dec. 17	5 grams	94	53	93	124	35
1928, June 28	0	101	73	118	156	28
1929, Jan. 11	0	128	84	132	190	28(14-4-10)

[1] Diets not absolutely accurate.

TABLE 9

Buddy H., Case No. 2084. Age at onset in June 1919, 3.2 years, weight 15.5 kg. which fell to 11 kg. in November 1920. Wgt. March 10, 1923, 11 kg. On December 31, 1928, weight 28.4 kg. hgt. 127 cm.

Date	Urine Sugar Grams	Diet C. Gms.	P. Gms.	F. Gms.	Alc. C.C.	Total Glucose	Insulin Units
1920, Dec. 1.	(3.0%)	75	51	58	15	110	
1920, Dec. 2.	17 grams	56	41	32	14	83	
1920, Dec. 7.	76 "	56	41	19	10	82	
1920, Dec. 14.	25 "	30	38	34	3	61	
1920, Dec. 19.	14 "	33	36	25	0	57	
1921, Feb. 16.	0 "	15	39	40	0	42	
1921, July 1.	0 "	44	41	40		72	
1922, Apr. 1.	0 "	26	28	39		46	
1923, Jan. 18.	0 "	24	37	32		49	
1923, Mar. 10.	0	27	38	37		53	
1923, Mar. 20.	0	37	41	58		67	2
1923, Apr. 19.	0	51	38	73		80	5 (3-0-2)
1924, Sept. 11.	0	50	42	80		82	11 (7-0-4)
1925, Dec. 1.	0	56	66	95		104	17 (13-0-4)
1926, Nov. 1.	0	56	66	95		104	17 (13-0-4)
1927, May 10.	0	70	71	100		121	30 (20-0-10)
1927, Aug. 5.	0	70	71	100		121	24 (18-0-6)
1928, Feb. 4.	0	75	71	100		126	27 (18-0-7-2)
1928, June 25.	0	90	72	86		140	29 (18-0-8-3)
1928, Aug. 30.	0	90	84	83		147	24 (18-0-6)
1928, Sept. 6.	0	100	85	90		158	24 (18-0-6)
1928, Nov. 15.	0	100	85	90		158	24 (18-0-6)
1928, Dec. 29.	0	110	85	90		168	27 (18-0-7-2)

Case No. 2560, Billy B. developed symptoms of diabetes in June 1921, at 5.1 years, but it was not diagnosed until March 1922 when the quantity of urine was 4000 c.c. and contained approximately 8 per cent of sugar and much diacetic acid. Wassermann negative. His grandfather is a diabetic, his mother most painstaking and B. B. at present appears to have one of the best records of any of my children. He was 12.3 years old, weight 33.9 kg. and height 57¼ inches and developing properly in August 1928, and at this time was taking carbohydrate 125 grams, protein 65 grams, and fat 125 grams with insulin 22 units. He is in good condition, but with less chance for exercise in the winter and while at home with a cold on December 18, 1928 his carbohydrate was 110 grams, protein 63 grams, fat 126 grams, insulin 32 units, but he was sugar-free and in fact had an insulin reaction. He leads his class at school and this brings up new queries in management which are deserving investigation. Between March 1922 and August 1928 his tolerance for carbohydrate has risen from 35 grams to 125 grams with 22 units of insulin.

Discussion: Before the use of insulin these five patients were all (save one and he nearly so) sugar-free and taking 58, 15, 30, 27, and 37 grams carbohydrate respectively. At the last recorded observation the carbohydrate was respectively 236, 166, 128, 110, and 125 grams. Similarly the total glucose before insulin was 91, 65, 71,

TABLE 10

Case No. 2560, Billy B. Onset at 5.1 years in June 1921
Weight March 1922 18.7 kilog. Height March 1922 120 cm.
Weight August 1928 33.9 kilog. Height August 1928 145 cm.

Date	Urine Glucose	Diet			Total Glucose Grams	Insulin Units
		C. Gms.	P. Gms.	F. Gms.		
1922, March 22	0	37	35	39	61	0
1923, March 18	0	40	44	82	74	4(2-0-2)
1924, March 3	0	55	95	74	118	14(8-0-6)
1925, Nov. 11	0	54	71	107	106	24(12-0-12)
1926, Dec. 10	0	63	66	107	112	24(12-0-12)
1927, Dec. 30	0	100	64	100	147	24(12-0-12)
1928, Aug. 2	0	125	65	125	175	22(12-0-10)

53, and 61 grams respectively, and at the recent observation respectively 313, 218, 190, 168, and 175 grams. Insulin is credited with the capacity of metabolizing 1.5 to 2 grams carbohydrate per unit and rather more than this, perhaps twice as much when a small number of units are administered. In our cases the gains in grams of carbohydrate per unit of insulin were 12, 6, 3.5, 3.1, and 4.0 grams respectively, and in total glucose were 15, 6, 4.3, 4.3, and 5.2 grams glucose per unit insulin. The total gain is not so impressive as the relatively more rapid gain after insulin had been used for a few years. Perhaps the attempt to raise the carbohydrate in the diet should have been made earlier.

Originally just prior to medication with insulin the calories per kilogram body weight were 26, 24, 83, 64, and 34 calories; at present they are 40, 34, 63, 56, and 56 calories. To some extent this meets Allen's contention that if total calories are kept constant the relative proportions of carbohydrate, protein, and fat can be shifted indiscriminately. In most of our instances the total calories per kilogram have been raised and that despite the fact that as children grow older their caloric needs grow less.

How much further should one attempt to raise the carbohydrate of these diabetics? I do not know, but already two have passed the 150 grams mark. I am interested to learn the experience of those who have advocated a high carbohydrate diet. In the meantime I shall forge ahead slowly and observe whether these children and other children can progress as have the two adults. There is no limit set by law to such an improvement.

Again I register the plea that others will record, as Brace and I have attempted, the course of some of their best patients.

CONCLUSIONS

1. The clinical course of cases of diabetes of prolonged duration does not show a tendency to increasing severity. Even the careless diabetic may do extraordinarily well.

2. The diabetics of prolonged duration die of the complications of diabetes rather than of diabetic coma which would be their more logical end

TABLE 11. GAINS IN TOLERANCE FOR CARBOHYDRATE, TOTAL GLUCOSE AND CALORIES OF FIVE DIABETIC PATIENTS WITH INSULIN DURING 6 OR MORE YEARS

Cases	Observation Preceding Treatment with Insulin			Interval	Last Observation			Gain in Tolerance with Insulin for		Insulin Units
	Carb.	Total Gluc.	Cal. per kilog.	Years	Carb.	Total Gluc.	Cal. per kilog.	Carb.	Total Gluc.	
359	58	91	26	6	236	313	40	178	222	15
632	15	65	24	7	166	218	34	151	153	26
1616	30	71	83	9	128	190	63	98	119	28
2084	27	53	54	6	110	168	56	83	115	27
2560	37	61	34	7	125	175	56	88	114	22

if they were approaching a stage of complete diabetes. Insulin refractory diabetics are conspicuous by their rarity in children. The diabetic mortality in children is approaching the vanishing point and in fact is decreasing for all diabetics under the age of 40 years.

3. My diabetic children daily challenge pessimism by their good looks, their vigor of mind and body and their normal sexual development.

4. Failure to demonstrate an increase in carbohydrate utilization by certain diabetics with insulin may be explained by the excessive calories allowed and the small amount of carbohydrate given over long periods.

5. Five cases of diabetes are reported by the author who show during periods of six years gradual gains in tolerance for carbohydrate varying from 88 to 178 grams with the use of 15 to 28 units of insulin. The gains in tolerance have been greater in the latter half of the period. There appears to be no reason why these gains should not continue.

Cardiovascular Lesions Following Injury to the Chest*

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THE superficial position of the heart and pericardium, directly behind the sternum and the adjacent cartilages, exposes them to danger from injuries to the anterior chest wall. With or without injury to the ribs, or even without obvious external bruising, an external blow may produce very serious damage to the intrathoracic structures. Corresponding to the injuries that take place to the lungs, liver, kidneys or spleen in serious acccidents, it may be plausibly surmised, the heart will suffer similarly the effects of sudden concussion or compression. The character of these effects will depend upon the status of the heart cycle at the moment of the accident, the resilience and resistance of the thoracic wall, and the form, momentum, and direction of the injuring force. The mechanical process in such accidents is subtle, of course, and the clinical picture often very difficult to solve. It is the milder form of accident that is the more commonly observed and offers real problems of diagnosis and therapy.

Thoracic injuries are generally classified into (1) those confined to the soft tissues; (2) those in which the ribs or sternum are involved; and (3) those which are complicated by injury to the lungs or the heart.

In the present paper, we shall consider the intrathoracic cardiovascular effects produced by direct or indirect violence from without. We emphasize this to make clear our usage of the term "traumatic injury" which we employ in our discussion. Literally, "trauma" means a wound; and its usage should be confined to those occurrences, in which physical violence or mechanical injury is involved, in the production of the lesion. Unfortunately, the word "trauma" has been used in a few published papers, in this connection, in the sense of bodily effort involving stress.[1] We believe that its employment in this way is not fully justified.

In cases of heart strain there is no wound produced in the usual meaning of the word. As a result of undue stress or tension, the lesion is produced from within the heart or aorta. Heart strain has been clearly defined as an organic condition associated with the sudden development of symptoms referable to the heart and aorta, which can be attributed to some unusual mus-

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[1]Hay, J. and Jones, H. W.: Trauma as a Cause of Auricular Fibrillation. Brit. Med. Jour., 1927, i, p. 559.

cular effort or over-exertion, and which results in physical disability of varying degree and duration.[2]

Particular attention also will be paid to the industrial aspects of non-penetrating injuries of the intrathoracic structures, and the considerations that arise in determining the compensation and liability as interpreted by various compensation laws.

The principal types of traumatic heart lesions that occur in consequence of direct and indirect violence may be classified under the following headings:

I. Direct Injuries
  A. Contusion and concussion of the thorax.
    1. Injury to the pericardium.
    2. Auricular fibrillation.
    3. Extrasystolic arrhythmia.
    4. Heart block.
    5. Injury to the valves of the heart
    6. Injury to the aorta.
    7. Injury to the heart musculature.
    8. Rupture of the heart.
    9. Miscellaneous types.
  B. Penetrating wounds.
    1. Fatal.
    2. With recovery and post-traumatic effects.

II. Indirect Injuries
  A. From without, such as falls with no direct injury to the chest.
  B. Heart strain.

[2]Kahn, M. H. and S.: Heart Strain in Its Industrial Aspects. Amer. Heart Jour., 1928, iii, 546.

Heart Strain and Its Consequences. Ann. Internal Med., 1928, 1, 790.

Inasmuch as pathological examination of the heart tissue in the milder form of accident to the thorax is not feasible, we can only consider these cases from the standpoint of analogous experimental studies It is only from the symptoms, and the clinical evidence of disturbance of rhythm, or other functional changes, that we can estimate the extent and determine the localization of the lesions. In severe cases, in which the clinical evidences are marked, or in cases that terminate fatally, an exact discovery of the damage produced is ofttimes obtained by autopsy.

Injuries to the thorax are common occurrences in various industries. This applies particularly to all those occupations in which objects are moved about, pulled forcefully out of piles, or thrown from one place to another. Being run over by a vehicle is one of the most frequent causes. An equally frequent accident is the crushing of the chest when a person is caught between two colliding vehicles, or between one such and a stationary object. A hazard also lies in unprotected straps around the wheels of machines. Falling directly to the ground as in tripping is a common cause of thoracic injury. Injury to the heart may also be produced indirectly, according to the natural laws of inertia, when one falls from a varying distance; in which case the force is applied directly to some part of the body other than the chest. Less frequent are the intrathoracic injuries produced by penetrating objects such as bullets, knives, or the sharp fragments of fractured ribs.

In some of our group of cases, the

mode of occurrence of the injury to the intrathoracic cardiovascular structures was described as follows:

A plank of lumber fell from a scaffold, striking the patient's chest.

While pulling at a bag on a wagon, its sudden release threw the man backward down to the ground.

Tripped and was thrown heavily forward to the ground.

Fell down five floors, striking the front of his chest against a pile of boards.

A two-ton machine fell upon the patient, throwing him supine.

While a heavy bag of coffee was forcefully pulled by means of a hook, it was suddenly released, hitting the patient in the precordium.

An automobile slipped off its "jacks" on to the patient's chest.

While standing, the patient fell from a bench, striking the front of his left chest against the floor.

The handle of a hand-truck struck the man across the sternum.

Was struck by a truck.

Struck in the chest by burglars.

Contusion and Concussion of the Thorax with Intrathoracic Injuries

A blow to the region of the heart may cause a contusion of the precordial tissues sufficient to extend to the parietal layer of the pericardium. If this is the full extent of the damage, the symptoms may be slight, and complete recovery may follow in a few days. Usually, the clinical effects of a contusion are temporary, with faintness, palpitation, and pain, which pass off in a few hours, or endure several days. In such cases, followed by recovery, no subsequent danger exists, and the patient may be considered well as soon as the effects have passed off.

But, in some cases, the effect of the injury is not localized to the mural tissues. As the thoracic wall is markedly yielding, a severe blow or sudden compression may momentarily alter the shape and volume of the intrathoracic space. Such a distortion may involve a displacement of the heart and mediastinum, and may be very severe, producing the symptoms of shock, and even resulting in death. This may happen with surprisingly slight, or even with no evidence of external injury to the thorax. The possibility of visceral injury should, therefore, always be kept in mind, even when the initial symptoms are mild in character. The particular evidences of cardiac affection, in such cases, lie in the development of arrhythmias, recurrent dyspnea, palpitation, and pain after exertion. Such cases come especially into notice when the questions of disability from work and liability for compensation are considered.

In severe cases, which are often spoken of as concussion of the chest, the patient may become unconscious and go into shock. The face is pale, the pulse weak and imperceptible, the respirations are shallow. Eventually a fatal issue results. Trauma in the region of the lower portion of the thorax, near the heart and diaphragm, is followed by greater shock than in more remote regions of the chest.[3]

[3]Crile, G. W., in Keen's Surgery, Volume V.

The prognosis in thoracic contusions depends upon the character of the injury. In cases associated with marked shock, the outlook is always grave.

The medico-legal questions involved in these cases often present no little difficulty for their satisfactory solution. To illustrate the obscure clinical effects often produced by direct violence to the chest, and the practical importance of these cases from the standpoint of the ensuing disability and the compensation liability, the following group of cases is briefly summarized:

In the following case, although cardiac damage would be anticipated from the severity of the accident, none was found. Disability due to cardiac injury was the patient's claim.

Louis H., a robust mechanic aged 45 years, was lying on his right side repairing the rear end of a heavy automobile bus. The latter slipped off the "jacks" supporting it, pinning down the patient's arms and chest. The bus was raised promptly and the patient released. He was not unconscious, was able to walk about, but felt a diffuse soreness across the front of the left chest, and pain along the right costochondral junctions. The next morning he was admitted to a hospital, where he remained abed for seven weeks with some improvement. X-ray at the hospital showed no evidence of fracture or pulmonary injury.

Eight months after the accident, he still felt soreness in his chest after straining, lifting or pulling, and even when at rest. Occasionally he had a sensation of tightness across the midsternal region and to the left side of the median line. He had no severe attacks of radiating pain, and no pain in the back of the chest or in the arms, and suffered no cough or other respiratory symptoms.

Physical examination, eight months after the accident, showed a heart rate of 86. The left border was within the nipple line, and the apex beat was distinctly visible in the epigastrium. Over the apex, and in the epigastrium, there was palpable a slight systolic thrill. The muscular quality of the first sound was fair. Over the pulmonic area, there was a faint systolic cardio-respiratory murmur. The basic sounds were not accentuated. The blood pressure was 156/90.

Summary—It is of particular significance in this case that there were no active symptoms referable to the heart or intrathoracic viscera immediately after the accident. It is presumable that if there were injuries to the heart as a result of the accident, immediate symptoms would have developed.

In this case, the chest was cushioned against the compressing weight by both arms. The thoracic wall yielded at the right costochondral junction as a result of the compression, producing localized pain along this line. The feeling of tightness and soreness across the chest that the patient felt after straining may have been due to the mural thoracic injury.

There is doubtless causal relationship between the accident the patient suffered and the symptoms he complained of.

In the following cases, injuries of varying severity to the chest occurred, but with no definite evidence of intrathoracic cardiovascular injury or aggravation.

The case recorded below portrays the obscurity of the effects of direct blunt violence to the front of the chest:

Madeline F., a little woman of 33, weighing only 82 lbs., slipped and fell forward, striking her breast bone against the corner of a table. She had immediate, very severe, localized, cramplike pain in this region, and

felt rapid palpitation, without irregular action. She went to bed immediately. The pain gradually lessened in severity, but there persisted for at least six months thereafter, a continuous sensation of heaviness and pressing pain in the midsternal region. Dyspnea and palpitation on exertion were also present. The disability was mainly attributed to the sternal pain.

Examination of the heart revealed a short, rough, rumbling systolic murmur localized over the apex, and another soft blowing murmur was heard to the left of the manubrium. There was extreme tenderness over the sternum and xyphoid, and adjacent left costal cartilages. Physical examination was otherwise negative. No fractures of the sternum or ribs were present, and x-ray of the heart revealed no significant findings. The electrocardiogram was equally uninformative.

Summary—Six months after the accident, we note, in this case, marked precordial and sternal tenderness, dyspnea, palpitation on exertion, an apical murmur, and disability due to sternal pain. It is difficult to say exactly what the pathological process was that produced the patient's symptoms and disability so long after the accident. But that small foci of mediastinal damage may develop in such a case is well recognized.

Jeremiah McC., an auto mechanic aged 55, was struck by the fender of a suddenly propelled truck in the region of the left lower ribs and left groin. When he regained consciousness, he found himself seated against a fence to which he had been carried. He had intense pain in the left lower abdomen, and an aching, numb sensation in the left hypochondriac region. There were no abrasions of the skin, no ecchymoses, and no bleeding from any part of the body. He had no cough or hemoptysis, no vomiting, melena or hematuria. He returned home by trolley, walking two blocks without assistance, and went to bed. That night the sharp localized pain in the left lower ribs became worse, especially on deep breathing, and there was some local tenderness. He felt dyspneic, especially in the recumbent posture, and had palpitation at times. Two days later, pleurisy was diagnosed.

One month after the accident, the patient had a sudden severe localized gripping pain over the left lower ribs, considerably below the region of the breast. He did not faint, but was relieved by hypodermic medication. Similar attacks of momentary pain recurred, not radiating to the arms, neck, or chest, with pallor and cold sweat, followed by palpitation. He had slight dyspnea and palpitation on moderate exertion, and was unable to breathe easily in the recumbent posture. The tenderness over the ribs finally disappeared.

Physical examination four months after the accident showed the left border of the heart just outside the nipple line, 10 cm. from the median line. The right border percussed 4 cm. from the median line. The apex beat was palpable in the fifth and sixth spaces in the nipple line. The sounds at the apex were good and the basic sounds were normal. There were no murmurs and no tender spots on the chest wall. There was an area of pain over the fifth and sixth left interspaces, corresponding to the region of the heart apex. The blood pressure was 122/70. There was no evidence of pleural affection. The electrocardiogram showed a rate of 66 and was otherwise negative.

One year after the accident, the patient appeared in good condition and had, for a length of time, not had any further attacks of pain or discomfort in the chest. He still claimed some disability on account of the shortness of breath, but clinical evidence did not indicate any further disability from the accident.

Summary—In this case, the injury produced apparently no permanent affection of the heart, although the symptoms and the attacks that the patient suffered from may be attributed to some direct trauma to the heart. The x-ray showed fibrotic phthisis in

the right upper lobe, but there was no evidence of aggravation of this condition.

James J. F., an old watchman, was attacked by burglars, beaten, bound, and gagged, and was struck in the left side of the chest. He was taken to the hospital with broken ribs and fractured knees.

Cardiovascular examination showed evidence of arteriosclerosis, with marked tortuosity, hardening, and calcification of the larger and smaller peripheral vessels. The heart presented effects of these arterial changes with frequent irregularities, due probably to auricular premature beats.

Summary — The arteriosclerotic process in this case was, of course, the result of senescence. It was important to decide, if possible, whether the assault in any way aggravated this condition. Obviously, it could not have affected the general arteriosclerosis. Whether the direct injury to the chest wall, producing fractured ribs, caused some myocardial damage is problematic.

Involvement of the Pericardium

The pericardium may react to a contusion or concussion of the chest with the development of acute pericarditis. This is often fibrinous in character, and often with slight effusion. Pericardial injury may develop following a sudden fall or lurch forward, in which the chest is struck against a projecting surface or edge. A blow to the chest by a missive or a fist, as in boxing, may be the history.

Generally, six to twelve hours intervene before an indication of pericardial involvement manifests itself by quite marked pain in the precordium and a slight rise in temperature, associated with difficulty and pain in breathing. The symptoms of traumatic pericarditis are not particularly different from the condition in its more usual clinical form. Important, however, are the history of direct injury, and the shorter duration of the clinical course in the typical or mild cases. Recovery may take place without pericardial adhesions. Often, however, the symptoms linger for a longer time, the patients complaining of some dyspnea and palpitation and tenderness over the precordium. In such cases, further unrecognized damage may be suspected.

Accidental traumatic pericarditis has to be differentiated from that form which occurs in an attack of sudden coronary closure or so-called stenocardia. In the latter, an aseptic local pericardial reaction takes place on the visceral pericardial surface, with the development of mild effusion, which also subsides within a short time. In such cases, however, the correlated advanced coronary lesion, and the symptoms of angina pectoria are part of the clinical picture.

Spitzmüller related the case of a man who fell from a height of about forty-five feet.[4] He was unconscious when picked up, and later complained of severe pain in the chest and abdomen. The pulse beat was small and rapid, but the heart was apparently regarded as normal in every other way. Toward evening, the patient began to vomit; his temperature rose slightly and the pulse increased to 170. The abdomen in the epigastric region was hard. The patient seemed to be

[4]Spitzmüller, W.: Injuries of Pericardium: Rare Case. Arch. f. klin. Chir., 1928, cl, 551.

in a state of shock. Laparotomy was performed, but no cause for the symptoms was found. There was very little free fluid and no exudate. Breathing continued difficult and painful. The patient now also complained of precordial pain radiating to the shoulder. Finally, on the third day, symptoms of pneumonia were manifest. They increased in severity rapidly and the patient died that night. At autopsy, a rent, 2.5 cm. long, was found in the apex of the pericardial sac—with no fluid in the sac itself. The neighboring organs were not injured by the fall.

The following case under our observation presents the question of pericardial involvement:

Edward A. K., aged 40, tripped and was thrown heavily forward to the ground. Later in the day, he felt some tightening in the chest and had a little difficulty in breathing. That evening he complained of shortness of breath, which became worse the next day. He remained in bed for a month after the diagnosis of acute pericarditis was made.

Physical examination a month later showed marked dyspnea, with a slow pulse of small volume. The heart sounds were somewhat muffled. There was no evidence of endocarditis and no symptoms or signs to suggest myocarditis. The lungs were negative, and the liver was not enlarged. About five months after the accident, cardiological examination showed the heart to be moderately enlarged with the right border 4½ cm., and the left border 11 cm. from the median line in the fifth space. The apex beat was not visible nor distinctly palpable, and was heard best in the fifth space inside the nipple line, at a regular rate of 78 per minute. The second aortic sound was slightly accentuated. The brachial arteries were somewhat tortuous, and the blood pressure was 140/95.

The electrocardiogram showed normal sinus rhythm, left ventricular preponderance, inversion of the *T* wave in lead I, and the *P* wave iso-electric in lead III. Teleroentgenogram showed the heart enlarged to the right and left, with definite hypertrophy of the left ventricle. The ascending aorta was not dilated.

The claimant resumed lighter work for five months or more before he died rather suddenly. He had not consulted a physician for these five months prior to his death.

Summary—There is definitely a history of an accident to the chest followed promptly by cardiac symptoms and the development of pericarditis. Whether or not the co-existence of acute coronary closure figured in its production must remain speculative. Five months after the accident, there was enough indication in the cardiac enlargement, hypertrophy of the left ventricle, dilatation of the ascending aorta, arteriosclerosis in the aorta, and changes in the electrocardiogram from which to conclude that there was some pre-existing affection of the heart. Yet, after the death of the claimant and from the available data, one cannot escape the suspicion that as a result of the accident, there was injury to the heart which produced pericarditis; and that it was not due to coronary involvement per se.

Auricular Fibrillation

It is well known that bodily effort or physical stress may produce auricular fibrillation.(1) In these cases, the onset in point of time is clearly defined, immediately following and definitely associated with unusual labor.

But the literature is very scant concerning instances of auricular fibrillation that follow direct violence to the chest. What the pathological changes are that produce this condition, one

cannot declare with certainty. However, the possibility of subepicardial ecchymosis in the auricular muscle must be considered. The cardiac damage in these cases is not disputable. The symptoms produced by its development are quite conspicuous, promptly causing more or less total disability. It is known that fibrillation diminishes greatly the cardiac output, as a result of which, symptoms of deficient cardiac function develop:[5] Dyspnea, palpitation, occasional sticking pain in the precordium or just below the left breast as well as the development of cough due to pulmonary stasis, and edema of the legs.

The accidental occurrence of auricular fibrillation will go on to spontaneous recovery in a short time, or to recovery following a period of rest and the use of special medication, or to recurrence with the persistence of the heart condition. In fact, once established, auricular fibrillation has a tendency to recur or even to become permanent.

Levison reports the case of a truck driver, aged 20, who was caught between two trucks and rather severely crushed in the thoracic region.[6] He was not rendered unconscious, and there were no fractures determined by the x-ray. There was no external wound, cut, or laceration, except a few discolorations from bruising. The patient complained of severe pain in the thorax generally, and of marked distress in the cardiac region with breathlessness. He was not cyanotic. Examination showed definite auricular fibrillation. The heart rate was 150 per minute, with marked pulse deficit. There was considerable tenderness over the chest region generally. The pupils of both eyes were dilated. The patient was weak, but clearly conscious and able to speak without any trouble. Deep breathing or any movement was difficult. The auricular fibrillation persisted for twenty-four hours. It is regrettable that an electrocardiogram was not secured during these first few hours. When it was taken thirty-six hours after the injury, it showed the P-R interval and the QRS complex normal, with slight sinus arrhythmia, and normal rate. The *T* waves in leads I, II, and III were abnormally large. There was definite left ventricular preponderance. The heart action seemed to be clinically normal at this time. The patient still complained of considerable pain in the region of the thorax and back, but the sense of cardiac distress and breathlessness had disappeared. When the patient had finally apparently recovered, physical examination of the heart revealed no evidence of any cardiac disease.

Illustrating this type of occurrence, is the following case which has been under our observation for some time:

Joseph G., a tall, well-nourished young carpenter aged 33, was struck across the front of his chest, as he was looking up, by a plank of lumber which fell on end from a building scaffold, 18 feet from the ground. He fell backward, momentarily dazed, stood up for a few minutes, feeling very weak, and leaning against the building for support. There was diffuse soreness in his chest and he "breathed hard as if all out of wind." He then continued lighter work with intervals of rest. When walking, he felt weak and dizzy, and stumbled

[5]Eyster and Swarthout: Quoted by Hay and Jones. Loc. cit.

[6]Levison, L. A.: Ann. Int. Med., 1927, i, 227.

in his gait. He worked half of the following day. During two weeks of hospital observation, he felt weak with pressing pain across the front of the chest.

Three months after the accident, his heart showed no enlargement and no murmurs, but showed rapid auricular fibrillation and a low blood pressure—104/78. There was no evidence in the history and in the physical examination of any previously existing heart condition. X-ray examination was also negative. The electrocardiogram showed coarse auricular fibrillation and tachycardia, but no ventricular preponderance and no indication of any pre-existing valvular disease. After a preliminary period of rest and digitalis medication, the patient was given three grains of quinidine sulphate every three hours for several days. There occurred quite remarkable improvement, with resumption of normal sinus rhythm. The symptoms promptly disappeared. The patient was able to walk about with progressive improvement and increasing strength, so that six months after the accident, recovery was quite complete.

Summary—In this case, cardiac symptoms, including the feeling of distress and soreness across the front of the chest, followed immediately upon the recorded accident. In point of time, the onset is therefore clearly and directly associated with the injury. We therefore believe that the condition of the patient had a direct relationship to the accident. There were not present in the history or physical examination any of the other causes which might induce auricular fibrillation, such as rheumatism, syphilis, arteriosclerosis, Grave's disease, or any other toxic causes. There was no valvular lesion, or cardiac changes incidental to a lesion, such as hypertrophy, accentuation of the basic sounds, or ventricular preponderance in the electrocardiogram.

The pathological changes that may have taken place from the injury are necessarily speculative. The possibility of subepicardial ecchymosis in the auricular muscle is to be considered. The process takes a long time to heal and therefore the auricular fibrillation may last an indefinite period of time.

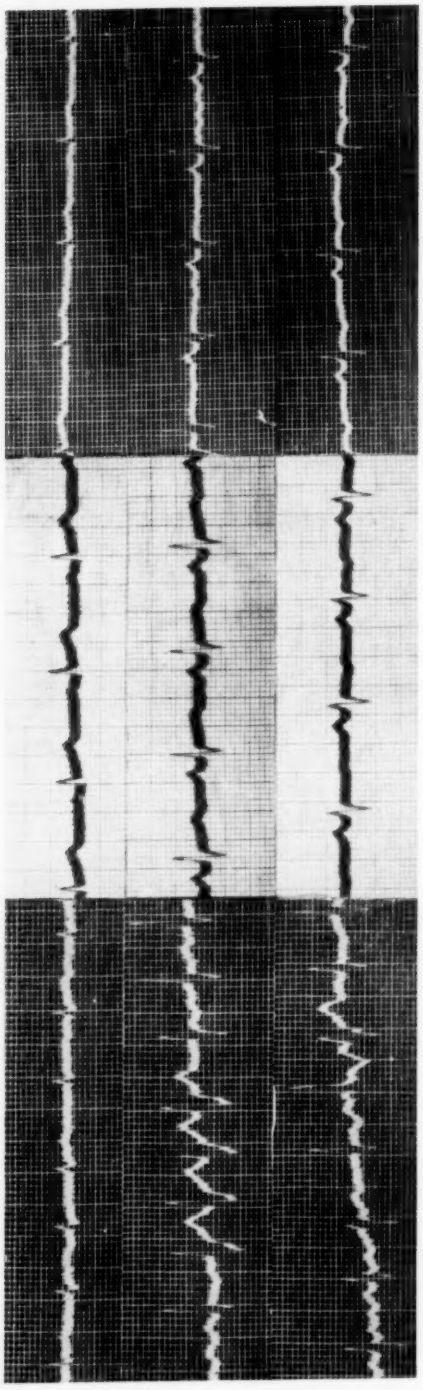
The electrocardiograms taken in this case are of interest. (See Fig. 1.)

During the preliminary period of observation, the electrocardiographic records showed rapid, coarse auricular fibrillation with an average rate of 150 per minute. There was an indication of left ventricular preponderance. Premature beats occurred singly or in groups, arising probably in the auricular wall in the region of the A-V node. Such a group is shown in lead II of the first portion of the tracing.

Two weeks after sinus rhythm had been restored and the effect of digitalis had worn off, the electrocardiogram showed a rate of 75 per minute. The left ventricular preponderance and the general voltage were, of course, unchanged. No premature beats occurred. The *T* wave was slightly taller in leads I and II, and was distinctly inverted in lead III. The P wave was rather sharp and there was a suggestion of its being notched.

A few months after the patient had been without medication, the electrocardiogram showed the *T* wave to be a little more inverted. This electrocardiogram is shown in the last portion of the record.

Patrick C. was a truckman, 63 years old. While straining to pull a 300 lb. case off his wagon by means of a hook, the latter



(1) Electrocardiogram three months after direct injury to the chest and heart. (2) Two weeks after the restoration of sinus rhythm. (3) Three months later, without any medication.

FIG. 1—Joseph G.

slipped out, and the claimant fell, striking his chest on the end of the wagon, and falling on his back to the ground five feet below. He was unconscious for a few minutes, but continued his work after that, delivering cases elsewhere. He then returned home, stayed in bed two days, worked one day thereafter, and then remained under the care of a physician. He became steadily worse and was admitted to the hospital four months later.

Examination at that time showed his heart to be enlarged. The apex beat, in the nipple region, was absolutely irregular, with auricular fibrillation. The muscular tone of systole was of poor quality. There was a loud blowing, systolic murmur at the apex and over the precordium. The radial, brachial, and temporal arteries were very hard and tortuous. The lungs showed a moderate amount of pleural effusion at both bases. This re-accumulated at intervals, necessitating frequent aspiration, with only temporary benefit. The auricular fibrillation persisted and the myocardial failure progressed, so that the patient died with pulmonary edema and advanced heart failure five months after the accident.

Summary—The patient showed arteriosclerotic myocardial disease and auricular fibrillation. If the fibrillation had existed prior to the injury, the man would have had some distress or inability to do his work. In the absence of this, we are compelled to attribute the onset of the fibrillation to the direct trauma to his chest that he suffered in the fall. Although no roentgenograms were available, clinically it appeared that there had been traumatic fracture of some ribs. The auricular fibrillation could very well have resulted from the accident, and death from the myocardial failure was correctly adjudicated the result of the aggravation of the heart condition, produced by the accident.

Mary McN. was a janitress, 39 years of age. She fell headlong down a flight of fourteen steps, striking the top of her head against the wall at the landing, and twisting her neck. She did not faint or become unconscious. The following day she felt sharp, shooting pain radiating from the xyphoid region up over the front of the chest and into the neck. The pain was aggravated by deep breathing, but not by moving about. Her family physician prescribed digitalis, and she remained abed for ten days.

Nine months after the accident, the patient complained of nervousness, fatigue, and exhaustion. She had rapid, severe palpitation, and a choking sensation on ordinary exertion, such as making beds or sweeping. There was also marked shortness of breath, especially on exertion. She had to sleep high on pillows to avoid a smothered feeling. She of course was unable to work.

Physical examination nine months after the accident showed the heart to be considerably enlarged to the right and left. The apex beat was forceful and palpable in the fifth space; there was also a palpable pulsation to the right of the lower sternum which was visible in the epigastrium. Over the apex, there was a marked systolic shock with a presystolic thrill. There was a short rumbling presystolic murmur audible over the apex, crescendo in character, preceding the systolic shock. The pulmonic second sound was accentuated.

During an examination one day, the heart action was absolutely irregular in force and rhythm due to auricular fibrillation. The rate was between 140 and 150 per minute. There was a suggestion of a presystolic thrill. The murmur was diastolic, short and blowing in character, and audible only during the longer pauses between heart beats. During re-examination on the following days, the heart action was forceful, and showed the presence of auricular premature beats, but no fibrillation.

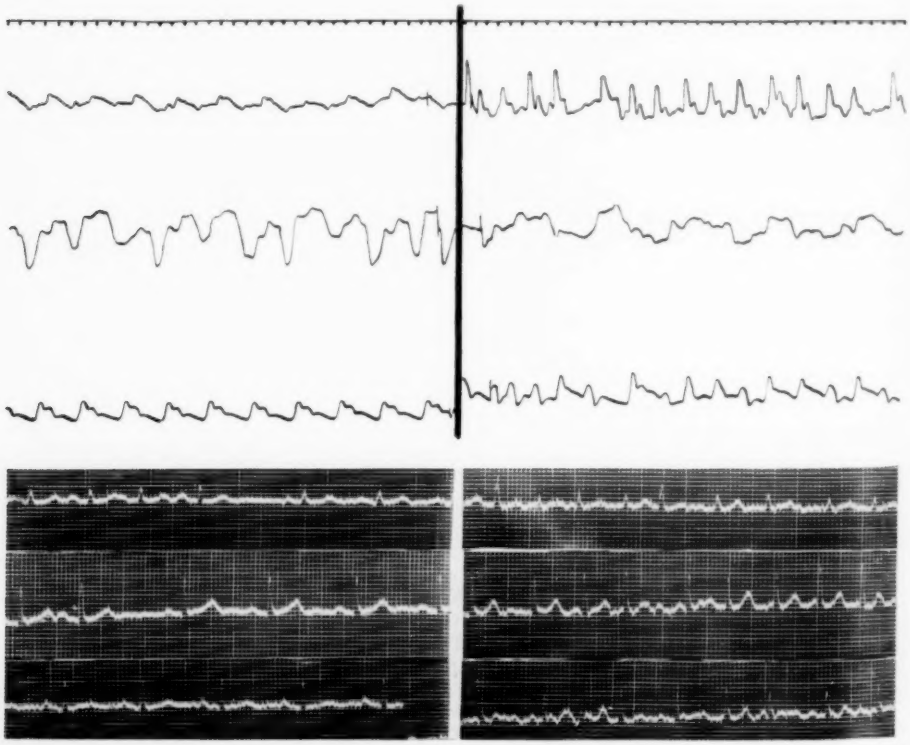
The blood pressure was 118/92 and 112/74 on two different occasions. The liver was slightly ptosed and enlarged, there was slight pretibial edema, and hyaline casts were present in the urine.

Teleroentgenographic studies showed the heart to be enlarged in its transverse and longitudinal diameters, and revealed the presence of mitral valvular disease.

Polygraphic tracings during the attack of fibrillation showed a rapid irregular radial pulse with the absence of the auricular wave in the jugular tracing. There was a suggestion of a presystolic thrill in the cardiogram. Another tracing, taken two days later, showed only the presystolic wave in the cardiogram due to mitral stenosis. (Fig. 2)

The first electrocardiogram showed very rapid auricular fibrillation and low voltage. There was also an occasional nodal premature beat. The *T* wave was upright in all three leads. The subsequent electrocardiograms showed slight sinus arrhythmia with the occurrence of auricular premature beats. The voltage was low and the *P* wave was notched and wide in lead II and inverted in lead III. There was no evidence of ventricular preponderance. (Fig. 2)

Summary—The patient had advanced chronic rheumatic mitral stenosis and regurgitation, with the occurrence of auricular premature beats and attacks of paroxysmal auricular fibrillation nine months after the accident when she first came under our observation. The heart was dilated and there was an old-standing congestion of the liver. The physical signs and the suggestive fact that digitalis medication had been prescribed promptly after the accident by the patient's family physician point to the previous existence of the valvular



(1) Polygraphic and electrocardiographic tracings showing normal sinus rhythm. (2) The same during an attack of paroxysmal auricular fibrillation.

FIG. 2—Mary McN.

lesion. That there was aggravation produced by the fall is suggested by the increased disability, the progressive orthopnea, the inability to climb stairs, and the enlargement of the liver and edema of the legs.

The natural progress of chronic rheumatic mitral stenosis and regurgitation eventually leads to symptoms and signs which would have produced the clinical picture found by us. The increase of her symptoms may have been due to such a gradual process. It thus becomes difficult to decide whether the alleged accident produced aggravation of her condition. There is no indication of any sudden and immediate heart affection resulting directly from the accident. There is no evidence that the patient developed an attack of auricular fibrillation soon after the accident; we therefore cannot attribute any direct relationship between this phenomenon and the patient's fall. The electrocardiogram shows no evidence of myocardial disease that might possibly be attributed to any trauma.

Permanent aggravation due to accident may result from a traumatic organic change in the heart. There was no evidence in this case of any direct violence to the chest. There is no evidence that the fall and fright produced immediate fibrillation from indirect effects.

The patient had temporary disability due to her accident on account of skeletal injuries. Her disability nine months later, however, was due to her chronic rheumatic heart disease, and not to any residual aggravation from the accident.

Extrasystolic Arrhythmia

Perhaps the most frequent form of irregularity of the heart action that takes place is that known as extrasystolic arrhythmia, in which premature beats interrupt the normal heart rhythm. These may arise in the auricle, or at any point in the ventricular musculature. The usual cause for their occurrence is focal myocardial irritation at one or more points, due either to rheumatic inflammation, or to coronary changes. A less common cause for their occurrence, however, lies in the lesions produced in the cardiac tissue by heart strain, by direct injury from blunt violence such as blows to the chest, or by indirect injury, as in falling from a height.

Where trauma is under consideration as a cause of this form of irregularity, it may be held liable by the process of exclusion. In young people, toxic and infectious causes have to be eliminated as possible factors. In older people, arteriosclerosis usually complicates the picture. It is, therefore, the clinical relationship between the accident and the development of this form of irregularity that guides us to the establishment of a causal association.

The symptoms of extrasystolic arrhythmia are often not pronounced. Usually, there is a sensation of a sudden interruption of the regular action of the heart, "as if the heart suddenly turns over," "a short thump in the chest followed by a pause," etc. There may be a feeling of momentary choking, faintness or dizziness, or darkness before the eyes, particularly if two or more extrasystoles follow in sequence. Extrasystolic impulses rarely give an

associated tinge of momentary pain. The occurrence of this form of irregularity of heart action and the locus of its origin in the cardiac musculature are determined by means of the polygraph and electrocardiograph.

Extrasystoles may recur for years, or they may subside after a short period of time. In older people with arteriosclerotic coronary changes, traumatic extrasystoles are more prone to persist than in young individuals. In some cases, quinidine sulphate produces beneficial effects. When extrasystoles are frequent, total disability may result. They often prevent an individual from doing strenuous work.

The following case serves as an example in which the consideration of this condition comes into play:

Gustav E. A., was a machinist and iron worker, 64 years old, who fell a distance of five or six floors (75 to 80 feet) upon a pile of boards, striking the front of his chest and fracturing several ribs. He was unconscious for a few minutes. There was no hemoptysis. Following the accident, he at times had pain in the back of his right chest. He had never fainted and had no anginal attacks. There was slight dyspnea and palpitation on exertion, and there developed slight edema of the legs.

Physical examination two years after the accident showed the heart slightly enlarged to the left. The sounds at the apex were clear. The aortic second sound was slightly accentuated. There were frequent ventricular premature beats and occasional short paroxysms of a series of six to ten premature beats which, by the electrocardiogram, were noted to arise at the apex of the right ventricle apparently at two different foci. The brachial and radial vessels were tortuous, and their walls thickened and sclerosed. The blood pressure was 194/120.

The electrocardiogram showed left ventricular preponderance. The *P* wave was diphasic in lead II. The *T* wave was low in lead I and isoelectric in leads II and III. (Fig. 3)

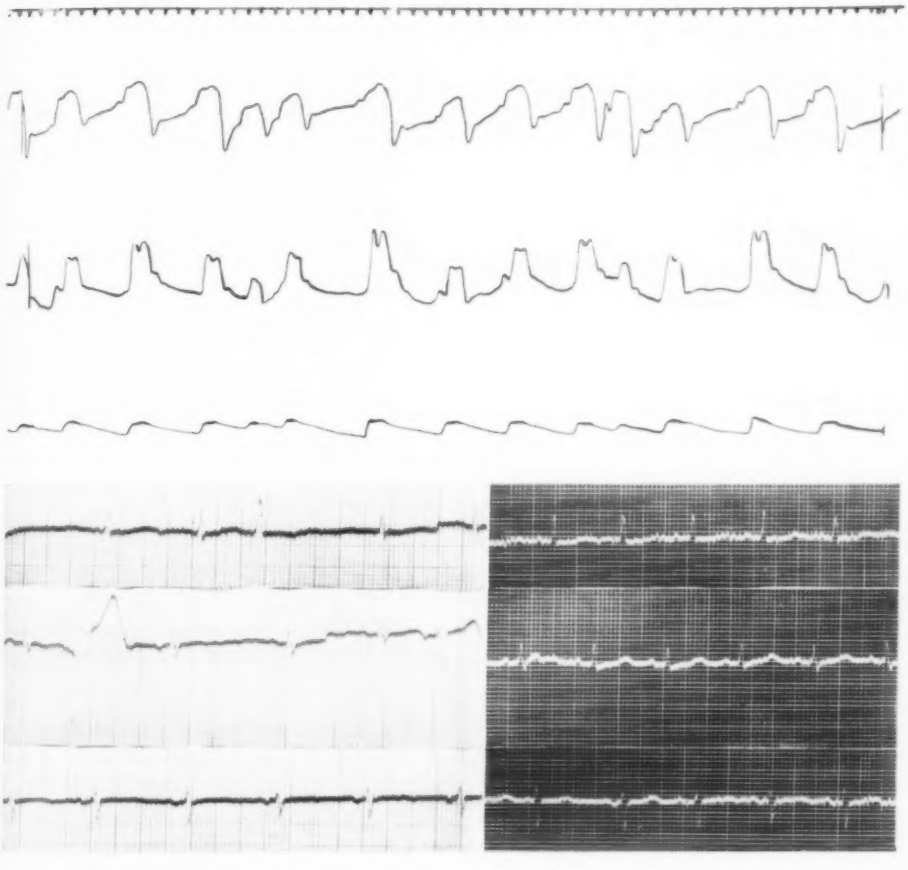
One year later, the patient became worse, showing increased pretibial edema. The heart became markedly enlarged. The apex beat was in the fifth and sixth spaces, outside the nipple line. The left border was within the anterior axillary line. The blood pressure was 192/100. No extrasystoles were audible. The electrocardiogram was little changed, but for the absence of extrasystoles.

Summary—The patient suffered from extrasystolic cardiac irregularity which was discovered after direct injury to his chest. The fact that he was able to do hard work previous to the injury indicates a previously good circulation. His arteriosclerosis was associated with his age and may have been contributory to the cardiac irregularity, or may even have been wholly responsible for it. Nevertheless, the severity of the direct injury to the chest makes it almost unreasonable to exclude direct trauma to the heart as a related cause of extrasystoles. No one can gainsay that the injury was a factor in causing the extrasystoles and the consequent, more or less permanent disability of the claimant.

Heart Block

The location of the trauma in the myocardial structure will predicate the functional disturbances or arrhythmia that may develop. Cases of heart block have been reported from direct injury to the chest. Of course, with penetrating wounds, the effect of an injury in the region of the bundle of His produces clinically what in the physiological laboratory is recognized as the Erlanger ligature. But the condition of heart block may follow trau-

(1) Polygraphic tracings showing the frequent extrasystoles, and trapezoidal cardiogram, and the presence of the "a" wave in the jugular curve.



(2) Electrocardiogram 2½ years after direct injury to his chest.

(3) The same 3½ years after the injury.

FIG. 3—Gustav E. A.

ma to the precordium without any penetrating wound, or even with no obvious injury to the thoracic wall.

Rosenson reports a case of temporary partial heart block in a boy aged 10½ years who had received a hard fist punch over the precordium.[7] Following the blow, he complained of weakness and a peculiar fluttering sensation over the region of the heart. He was unable to walk, felt faint and was carried to bed. Two days later, on attempting to walk, he fell and was unconscious for a few minutes. Two weeks after the injury to his chest, he still suffered slight dyspnea on exertion. The heart showed no enlargement. There was a pulmonic systolic murmur, functional in character. The sinus rate was 120. The electrocardiogram showed varying grades of

[7]ROSENSON, W.: Heart Block in a Child Following Trauma to the Precordium. Amer. Jour. Dis. Children, 1924, xxviii, 594.

heart block with a rapid auricular rate. The *T* wave was inverted in lead III. The conduction time was 0.28 of a second. Within a month after the accident, with rest in bed, the child made a complete recovery. The electrocardiogram indicated a return to normal rhythm.

The pathological process that developed in the conduction system following the blow to the chest wall is conjectural. It may have been a slight hemorrhage in or about the node of Tawara, or a functional change in the intracardiac nervous mechanism.

Injury to the Valves of the Heart

Injury to the front of the chest, with or without injury to the ribs, or even without obvious external bruising, may cause rupture of one of the valves in the heart. The causes noted are blows on the front of the chest, compression between wagons, kicks, and falls from a height. With the increase of automobile casualties, valvular injury is not infrequently the result of accidents in traffic.

Rupture of an aortic or mitral valve may be produced experimentally by smiting the walls of the thorax in animals, or in the cadaver.[8] In the reported experiment on the cadaver by Potain and his co-workers, artificial hydrostatic pressure through the aorta caused rupture of the aortic cusps with a pressure varying from 116 mm. Hg. to 484 mm. Hg.[9] Artificial hydrostatic pressure in the left ventricle produced rupture of the chordae tendineae by a pressure of 1050 mm. (1½ atmospheres), and of the flaps by 184 mm., and heart rupture by 896 mm. After the aorta was filled with water to give normal blood pressure, a sudden blow on the upper chest produced tears of the aortic cusps. The force of the blow raised the aortic blood pressure from the normal 120 or 150 to 170.

These lesions have been known to occur clinically in consequence of external injury to the thorax. This may occur in a heart and valves altered by disease, as well as in valves previously healthy. Senac, in 1778, was the first to discuss traumatic rupture of the valves, and he cited three cases.[10] Thirty years later, Corvisart amplified our knowledge of the subject; and since then a great number of clinical and pathological reports has accumulated.[11]

In the mechanism of such an accident, the external blow violently compresses the thoracic cage. The glottis may be closed in deep respiration, and the thorax held rigid. We may conceive of a violent concussion of the distended aorta just at the moment of full ventricular systole. Under such circumstances, rupture of an aortic cusp might take place. In cases of rupture of the mitral valve, on the other hand, the blow must have fallen upon the heart in diastole, with the ventricle filled with blood. The sudden increase in ventricular pressure produced against the closed and re-

[8]Potain: Clin. Med. de la Charite, Paris, 1894.

[9]Barie: Recherches sur les Ruptures Valvulares du Coeur Rev. de Med., 1881, i, 132.

[10]Senac: Traite des Maladies du Coeur. 1778. Vol. I.

[11]Corvisart: Essai sur les Maladies du Coeur. 1811.

sisting, thin mitral valve may tear it across or away from the chordae tendineae, or even from the papillary muscles. There are cases reported in which simultaneous rupture by violence was found to have involved both valves.

In the history of some of these accidents, the distinction between outer and inner stress can be made only with difficulty. Thus, when an individual, like in the case recorded below, resists the attack of burglars who have pinned his arms and beaten his chest with blows from their fists, he may have sustained heart injuries from the direct violence, as well as from the stressful effort of his resistance. But, probably the mechanical process of the production of injury is similar in both cases, force exerting itself from the suddenly increased pressure in the aorta against a filling and stretching left ventricle.[2]

Rupture of a cusp of the aortic valve is much more common than of the mitral valve in cases of direct trauma to the anterior chest wall. Perhaps one of the reasons lies in the anterior position of the aorta, which renders it more exposed to the effect of direct concussion. Peacock collected 17 cases in which one or more of the aortic crescents was ruptured in this way.[12] Wilks recorded the case of a youth of 19 in which a blow to the chest, an accident such as might happen in boxing, ruptured the posterior cusp of the aortic value from its free margin to its base.[13] In Daganello's case, a healthy man was crushed between a cartwheel and a post.[14] He continued work the same day, but the next day he was unable to exert effort without the onset of dyspnea. Little by little, he grew worse, with cyanosis and pulmonary stasis. There was also an aortic diastolic murmur. Autopsy, six months later, revealed dilatation of both cardiac cavities and hypertrophy of the left ventricle. The healthy aortic cusps were found completely torn away from a healthy aortic wall. Colwell and Mark published the case of a man, aged 20, who fell from a scaffold three weeks after he had a negative physical examination of his heart.[15] On re-examination ten days after the accident, he had a double aortic murmur, Corrigan pulse, and enlargement of his heart toward the left.

Barie gives thirty-eight cases of traumatic aortic injury.[9] He records thirteen cases of rupture of the valves of the heart by external violence without external wound, including eight cases of rupture of the aortic cusps and three of the mitral valve. As some of these cases are very typical, they are briefly abstracted here:

> 1. A healthy laborer 45 years old fell a distance of 15 feet, striking his left chest against a hard pavement. There was ecchymosis of this region for sev-

[12]Peacock, T. B.: On Some Causes and Effects of Valvular Disease of the Heart. J. Churchill & Sons, London, 1865. Page 34.

[13]Wilks, S.: Trans. Path. Soc., London, 1865, xvi, 77.

[14]Deganello: Arch. Mal. du Coeur, Paris. 1908, i, 710.

[15]Colwell and Mark: Rupture of the Aortic Valve. Brit. Med. Jour., 1901, ii, 1736.

eral days, but no fracture of the ribs. He was disabled and in bed. Eleven days after the accident, he developed severe precordial pain and palpitation. Dyspnea developed and increased, and within two months edema of the legs developed. The heart became hypertrophied, its action heaving, with a very loud diastolic aortic murmur. The influence of the traumatism in the production of the lesion in this case is unquestionable. The trauma produced rupture of one or more of the aortic cusps.

2. A healthy man of 35 was struck in the left chest by the head of a comrade. He lost consciousness immediately, and soon began to feel a constant oppression in the chest, dizziness on even slight exertion, dyspnea, and palpitation. He developed marked signs of aortic insufficiency.

3. A boy of twelve fell from his horse, landing with outstretched arms on the ground. He developed signs of cardiac disease and died with cardiac failure. At autopsy, a distinct notch was found in one of the aortic cusps, the location of a tear resulting from the accident.

Heidenhain reports the case of a healthy sailor, 49 years old, who was struck in the chest by an anchor which he was hoisting.[16] He immediately had severe pain in the chest, but continued at moderate work for one week. The pain persisted, dyspnea and palpitation after exertion developed, and after six weeks, the patient took to his bed. At times, the attacks of palpitation occurred even during rest. At that time the heart was considerably enlarged both to the left and right, with epigastric pulsation and mild arrhythmia. A loud systolic murmur and a soft diastolic blow were heard over the aortic area and also at the apex. The diagnosis was made of aortic stenosis and regurgitation. In this case, the sailor suffered no previous cardiac condition, as his work had always been so strenuous. The likelihood of a traumatic aortic lesion produced by contusion of the chest was accepted by the author.

Peacock reports the case of a previously healthy dock labourer, age 36, who was pulling with other men at a sugar hogshead; his hand slipped and struck him a severe blow on the left side of the chest, and he fell backwards.[12] He immediately felt severe pain in the region of the heart and became faint, and in the evening his breathing became difficult. These symptoms subsided in a few days, but never entirely ceased. He afterwards got worse till the time at which he was admitted into the hospital. He then had marked symptoms of cardiac disease, a double murmur was heard over the aortic orifice, the pulse was characteristically regurgitant, and he was dropsical. He died three and a half months after the accident. The heart was found to be greatly enlarged. The left angle of the posterior semilunar segment at the aortic orifice was found to be torn from its attachment, so that it was quite loose and readily admitted of retroversion, allowing free regurgi-

[16] Heidenhain, L.: Deutsch. Ztschr. f. Chir., 1895, xli, 286.

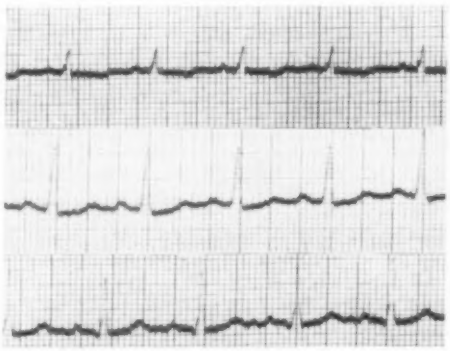
tation from the artery into the ventricle.

In this category of aortic valvular lesions following direct injury to the chest belong the interesting two cases that are reported below:

John O., a robust fellow aged 26, had been working as a laborer for some years. One afternoon, while he was supporting with his out-stretched arms a two-ton platform standing on edge on the floor, it fell over toward him. He was thrown supine, the weight of the platform falling against his chest. It was raised and the patient pulled away from under it unconscious; he so remained lying on the floor for about five minutes. When he roused, he felt severe aching pain across the front of the chest and in the epigastrium. He also had difficulty in breathing, but suffered no palpitation, cough, hemoptysis, or dizziness at that time. He was then taken by ambulance to a hospital.

The chest showed no deformities, no evidence of external injury, and no ecchymosis. There was diffuse tenderness over the midsternal region, the second and third left ribs, and also over the xyphoid region. No fractures had taken place.

Physical examination three months after the accident showed the heart to be markedly enlarged to the left and right. The left border was 12 cm. from the median line, outside the nipple line; the right border was 7 cm. from the median line in the second, third, and fourth spaces. The apex beat was heaving, in the fifth space below the left nipple. Over the apex and precordium, radiating upward to the aortic area and toward the vessels of the neck, there was a loud blowing systolic murmur replacing in good part the first heart sound. Over the base there was heard a short diastolic blowing murmur. There was present a Corrigan radial pulse and also a visible capillary pulse. Over the femoral artery there was heard the pistol-shot sound of ventricular systole and just preceding this was another systolic sound. There was visible brachial pulsation with evidence of slight brachial and moderate temporal tortuosity. The brachial blood pressure was 140/34, while the popliteal blood pressure was 180 systolic and heard faintly to 250; the diastolic pressure sound was heard at zero. The electrocardiogram showed no ventricular preponderance.



Electrocardiogram three months after direct injury to his chest and heart.
FIG. 4—John O.

Radiographic examination revealed some pulmonary congestion on both sides. The heart was horizontal in position, enlarged to the right and left. The left ventricle was rounded and prominent. The retrocardiac space showed a normal descending aorta. There was no definite evidence of enlargement of the left auricle, as demonstrated by contrast-filling of the esophagus. The findings pointed to enlargement of the left ventricle and right auricle and suggested enlargement of the right ventricle.

The blood Wassermann reaction was *four plus*.

The subsequent progress of the case was very rapid. Pain and distressing pressure across the chest became more and more marked, compelling the patient to sit up almost all day and all night. Frequent attacks of cardiac asthma developed with enlargement of the liver, congestion of the pulmonary bases, and progressive edema of the legs. The mild antiluetic treatment that was instituted was not of any help and, from the observation, did not contribute to the sudden death which occurred four and a half months after the accident. Unfortunately, no autopsy was obtained.

Summary—From the history of the severity of the injury in the accident, one is compelled to adduce a causal relationship between it and the condition of aortic regurgitation ending in cardiac failure and death. Because of the four plus Wassermann reaction, we cannot be certain in this case that the heart was normal before the accident. A previously diseased valve is more susceptible to traumatic effects than a sound valve.

Alfred B., a chauffeur aged 37, was inside a truck, unloading it of bags of coffee. After forcefully pulling by means of a hook at a bag of coffee weighing 150 lb., it was suddenly released, hitting him across the left side of the front of the chest and throwing him back against the side of the truck. For a moment he felt breathless: "the blow took the wind away." He rested, standing, for about five minutes, "feeling winded," and felt a slight localized pain along the lower front of the left chest, slight dizziness, and blurring of vision. He did not feel faint or nauseated, and did not cough or expectorate blood. He continued to unload lighter bags, and then went home by subway, and up two flights of stairs slowly, feeling the pain which became slightly more intense, together with frequent blurring of vision and dizziness. On walking upstairs, he felt dyspneic, with increased pain and palpitation. He continued at hard work for the following six days, obtaining some help in unloading. During this time, aching pain below the left breast region was continuous; at times, it became momentarily worse, compelling him to draw the truck over to the curb. The pain and inability to draw a satisfying breath persisted, with dyspnea and palpitation on exertion. At times, he awoke during the night, distressed for breathing for a few minutes. There were no anginal attacks.

Physical examination a year after the accident showed very slight prominence of the precordium. The apex beat was faintly seen, diffusely felt in the fifth space and at the sixth rib, four inches to the left of the median line. The heart borders indicated considerable enlargement both to the right and left. Over the apex, there was a loud blowing systolic murmur, slightly heard toward the left axilla, and faintly in the left interscapular space. Over the aortic area, this murmur was louder and rougher in character, transmitted upward. The pulmonic second sound was louder than the aortic. The basic sounds were not accentuated. There were tender spots over the second left rib, four inches from the median line, and over the sixth left rib, four inches from the midline. The blood pressure was 142/90.

A teleroentgenogram of the heart and aorta showed definite prominence of the cardiac shadow to the right. The aorta was slightly widened.

Summary—The patient had definite involvement of the aortic valve, and also involvement of the mitral flap, both on an atheromatous basis. This condition pre-existed for a long time and was not related to the injury. It is, however, definite that following the blow to the chest, the patient developed immediate symptoms which had not been present prior to the injury, and which persisted for a long time after. We must, therefore, assume that there did not occur some direct effect upon the heart, producing the pain and other symptoms. Usually, in such cases, the physical signs are either absent or very slight.

Cardiac injury, with the presence of ecchymosis in the heart muscle, takes a long time to heal, and healing is expedited by rest and avoidance of physical strain. Of course, symptoms due to aortic atheroma may often develop spontaneously and confuse the later analysis of the case.

The most important clinical characteristic of these cases is the immediate

development of physical signs referable to the valvular lesion. Immediately after the injury, the patient may be dazed or unconscious. Dyspnea, palpitation, and pain are prompt in their development, with weakness, dizziness, and often hemoptysis. The pain may be referred to the epigastrium, sternum, submammary region, or between the shoulder blades. In aortic valve injury, the patients sometimes feel a sudden tearing of the tissue in the chest. Sudden momentary faintness and dizziness occur, which become worse on exertion. These may be associated with palpitation, cardiac asthma, and extrasystolic arrhythmia. Cyanosis and edema usually develop, either promptly or later. A loud, rough, musical murmur is audible over the entire precordium and often at a distance of one to three cm. from the surface of the chest. In traumatic aortic valvular lesions, the patient is immediately totally disabled from his work. In some cases, the patient has continued lighter work for a short time, while suffering pain.

Although rupture of the mitral valve or chordae tendineae has been produced experimentally in the cadaver, clinical occurrence of this lesion with survival of the patient is not very common. Neither is it as well substantiated in the literature as cases of aortic lesions. However, the mechanism of its occurrence is readily appreciated from the theoretical discussion presented above. This lesion is not produced by indirect injury to the heart, such as occurs when an individual falls from a height, landing on his pelvis. Of course, if the valve was previously diseased, a severe injury may cause additional changes. Direct violence to the anterior chest, however, is a rare cause of a mitral valve rupture. More frequently the clinical effect that must be considered in these forms of accident is the aggravation of a previously existing mitral lesion. To illustrate this, the following case reported by Barie is presented:[9]

. A woman 56 years old was able to pursue her occupation as a manageress although she had a well-developed mitral regurgitation. One day at work, she fell eight feet down a stairway, violently striking the region of her left breast against the banister. She lost consciousness and spit some blood ten minutes after the accident. That day she developed sharp precordial pain, and a sensation of smothering and palpitation. The heart was enlarged. Edema, orthopnea, and cyanosis developed in a month. The pulse became small and irregular. The systolic murmur became rough and prolonged. She soon died with pulmonary congestion. Autopsy showed rupture of four of the chordae tendineae of one of the mitral cusps. In this case, aggravation was produced by rupture of the chordae tendineae, and the regurgitation was increased.

It is much less easily to be granted that mitral stenosis and aortic stenosis can arise directly from similar causes. That stenotic lesions of the aortic and mitral valves following direct violence to the chest do occur has been asserted, and cases have been reported in the literature. Thus Allbutt reports a case of mitral stenosis in which, after close inquiry, he confidently attributed the lesion to the kick of a horse on the

cardiac area.[17] The patient was a young man, aged 18, and the symptoms were some months in declaring themselves, yet the causal relationship seemed to him conclusive.

The mechanism of this has been a matter of speculation. Experimentally, Rosenbach produced rupture of the aortic cusps by means of a sound introduced through the carotid artery.[18] He found thrombosis at the site of the lesion. He then introduced infectious material which created an endocardial inflammation with stenosis in the region of the injured valve. It has been therefore assumed that following injury to a valve, stenosis may be produced by superimposed infection. Litten and von Leyden likewise believed that minute hemorrhages or superficial laceration following trauma may be the foci for further endocardial pathology, and that thrombotic vegetation may supervene at these points.[19,20]

Reubold and Rose consider the stenosis in such cases produced by extravasations of blood which organize and mechanically distort the cusps.[21] The valvulitis of traumatic endocarditis is due to contusion. It is not of the character of verrucous affection and differs pathologically from the infectious type of inflammation. It must be mentioned that a traumatically injured valve is vulnerable to subsequent rheumatic infection. Thus the inflammatory process that develops over the wound in a valve may lead to shrinking or scarring with the adhesion of the valve fragments.

The physical signs can furnish us data to indicate whether the mitral or aortic valve is the one affected as a result of the accident. But we have no way of asserting with surety the exact nature of the lesion. We can only repeat with Laennec that the exact type or amount of rupture may only be suspected; it is impossible to determine this from physical signs.

The duration of life following traumatic rupture of a valve is very variable. Immediate death may occur. Temporary recovery may be sufficient to allow for the resumption of light work. Even healing may occur. Prognosis is better in cases with pre-existing valvular regurgitation as in these, the heart has already undergone compensating hypertrophy.

In general, mitral rupture is more often promptly fatal than aortic rupture. In aortic cases, life may be prolonged for several months, but the prognosis is always grave. Traumatic aortic valvular lesions are graver as to life prognosis than the infectious aortic lesions.[22] Laennec asserts that tear of the chordae tendineae is less soon fatal than tear of the valve flap itself.[23]

[17]Allbutt, C.: Transac. Clin. Soc., ondon, 1873, vi, 101.

[18]Rosenbach: Arch. f. Exper. Path. u. Pharmak., 1878, ix, i.

[19]Litten: Ueber traumatische Endocarditis. Centralbl. inn. Med., 1901, xxii, 513.

[20]Von Leyden: Rupture der Aortenklappen. Berl. klin. Wchnschrft. 1889, xxvi. Idem 1892, xxix.

[21]Rose: Deutsch. Ztschr. f. Chir., 1885, xx, 319.

[22]Foster: Med. Times and Gazette, 1873, ii, 657.

[23]Laennec: Treatise on Diseases of the Chest and Mediate Auscultation. Translated by John Forbes, London, 1834.

Injuries to the Aorta

Besides injury to the aortic cusp, the wall of the vessel frequently suffers in accidents due to direct violence to the front of the chest. A blow on the chest, a sudden fall, or the jar of an accident may cause rupture of the aorta with the formation of a dissecting aneurysm. This has been recognized since the time of Vesalius. Usually, the cases present the symptoms of ordinary saccular aneurysm of the aorta, but in one case recorded by Allbutt, there was dilatation of the aorta. In aneurysm of the abdominal aorta, trauma is a very common etiological factor. The essential pathology consists of a split of the intima and inner portion of the media; a dissecting aneurysm may follow with healing or with a rupture; a saccular aneurysm may form, or the wall of the aorta may rupture completely.

Allbutt reports two cases which instance the formation of traumatic aortic aneurysm:[24]

Mr. B. was a well made man under forty, whose health was excellent. One day, on endeavoring to arrest a carter, the man backed his horse in such a way as to jam B. between the cart-tail and a stone wall. He was seized at once with intense pain and breathlessness, and soon after undoubted symptoms of aneurysm of the ascending aorta were developed.

Dr. Moxon published a very similar case.[25]

In another case, aneurysm of the descending aorta was caused by a machinery accident. The patient, a well-made and previously healthy man, was whirled off his feet by a gearing strap, and before the engine could be stopped, his chest was caught between the strap and a wheel and was seriously crushed. He suffered intensely from pain and dyspnea; and soon after symptoms of an aneurysm to the left of the sternum were developed.

In all these cases a sudden and violent compression of the systemic arteries distends the aorta, at a time, perhaps, of cardiac systole—the brittle inner coat gives way, the blood gradually distends the elastic coats, the vasal nerves are palsied, a sac forms, more nerves are palsied, and vasa vasorum are occluded; so that widespread mischief is thus added to the original injury.

Kemp reported a case of traumatic rupture of the aorta in a patient who was struck on the chest by a portion of a stone flywheel, which had separated while revolving at full speed.[26] He had a large abrasion over the sternum. The post-mortem findings in the thorax were: an extravasation of blood into the subcutaneous tissues, and underlying the pectoralis major, an extravasation of the blood into the anterior mediastinum. The sternum was fractured obliquely from above downward and backward at the level of the third and fourth costal cartilages. The lungs were deeply congested. The pericardium was greatly distended with blood which had escaped from a transverse tear of the

[24] Allbutt, C.: Effects of Overwork and Strain on the Heart and Great Blood Vessels—St. George's Hosp. Reports, 1870, v, 23.

[25] Moxon: Med. Times and Gazette, 1870, vol. 2, page 95.

[26] Kemp: Lancet, 1923, ii, 953.

aorta, just beyond the line of the aortic valve. The exuded serum was under considerable pressure.

In McWeeney's cases, the aortic arch and the left pulmonary artery beneath was torn across three-quarters of an inch beyond the left subclavian by a fall from a scaffold.[27]

McFadzean reports the case of an old man who died suddenly one morning when attempting to rise from bed.[28] Necropsy showed the presence of four aneurysms occupying the whole extent of the abdominal aorta and the greater part of the external and common iliac arteries. All the large arteries of the abdomen, from the origin of the celiac axis to the termination of the external iliac arteries on either side, were degenerated, calcified, irregularly dilated and aneurysmal. The patient had been an acrobat, a strong man and trapeze artist. Almost every day, for twenty years, he spent some time swinging and turning on a hard trapeze and rolling round the instrument on his stomach. This may have been a probable cause of the extraordinary aneurysmal condition of those vessels which, in the process of his acrobatics, would be most exposed to injury. There were no postmortem signs of syphilis.

Injury to the Heart Musculature

A number of cases in the literature indicate strikingly that due directly to external violence, small foci of myocardial damage may develop. Cases reported by Fisher[29] and Reubold[30] were each concerned with a serious accident, in which severe trauma took place without any penetrating wounds or rib fractures in the region of the heart. At autopsy, the pericardium was found torn and there were only superficial wounds of the myocardium. Death in these cases was due either to concomitant injuries to other parts of the body, or to hemorrhage into the pericardium from the superficial myocardial wounds.

A diagnosis of myocardial injury in such cases, if feasible, is important as surgical measures might be curative. But when other parts of the body are injured simultaneously the diagnosis is exceedingly difficult.

Whenever a colliding force has been directed against the precordial region of a patient, the possibility of myocardial damage must be kept in mind. If fracture of the ribs or injury to the pleura occurred such suspicion becomes emphasized. The presence of irregular action which supervenes upon violence to the heart is a significant sign. Although the pain has no particular characteristics, it is usually localized over the injured area and sometimes radiates to the left axilla or below the left breast. In most cases of myocardial damage due to external violence, the signs of a transitory pericarditis develop. Usually the heart sounds are lessened in volume

[27]McWeeney: Brit. Med. Jour., 1903, i, 251.

[28]McFadzean: Brit. Med. Jour., 1928, ii, 154.

[29]Fischer: Die Wunden des Herzens und Herzbeutels. Von Langenbeck's Arch. f. klin, Chir. 1868, ix, 571.

[30]Reubold: Bemerk. uber die Quetschung. Friedrisch's Blätter f. gerichtl. Med., 1890, xli, 285.

and deficient in muscular quality, although no murmurs need be present. Particularly significant is the persistence of a rapid heart rate in the absence of fever. The irregularity of the pulse and its variability of rate are very important.

It must emphatically be recognized that milder injuries of the myocardium may take place consistent with life over a varying length of time. The symptoms and signs, as well as the disability in such cases, usually persists.

Direct violence to the chest may, of course, act like a previous disease in pre-disposing the heart of a person to the effects of heart strain. Usually, however, the resulting condition must be attributed to the direct violence rather than to the strain, for the reason that the aggravation of symptoms by effort usually develops within a short time after the violence. Thus, in a case of Schuster's, a healthy laborer, 43 years old, received a blow in the chest by the harness bar of a wagon, and was thrown back to the ground.[31] He immediately recovered, except for some pain which persisted for two weeks. At that time, following a hard strain of lifting, he developed a sudden and severe epigastric pain radiating around to the chest and back, with a choking sensation. The patient threw himself screaming to the ground in extreme anxiety. There was marked epigastric tenderness, but the pulse rate and heart appeared normal. The following day, large doses of morphine produced some relief, but he was still pale, cold, cyanotic, and the heart action and tone were weak. The pulse was very small and of normal rate. He died suddenly after a few hours. Autopsy showed hemorrhage into the anterior and posterior mediastinum and also into the pericardium. Under the epicardium of the right auricle there were a number of hemorrhages. One of these encircled the coronary-artery and vein, encroaching on their circulation There were also hemorrhages under the endocardium of the right auricle. Extravasations of this kind were also present in the muscular substance of the left ventricle as well as the endo- and epicardium. The aortic wall was torn horizontally just below the semilunar ring. This tear involved the intima and media and a small dissecting aneurysm was present. There was no pathological process of any kind in this region.

In this case, it is amazing that with such extensive injury to the heart, muscle and aorta, the patient was able to do even strenuous work for a time and felt only moderate pain. The rise in blood pressure, entailed by the additional lifting two weeks after the original accident, apparently produced the severe symptoms and total disability that terminated in his death. In such cases, the aggravation produced by strain was secondary. The essential liability falls upon the direct injury by violence.

We may include the following interesting case of ours in this group:

Christino G. aged 45 had been pushing a hand truck for two years without pain or discomfort. One day, a very heavy load

[31] SCHUSTER: Ztsch. f. Heilkunde, 1880, i, 417.

falling from a height weighted the far end of the hand-truck, catapulting the handle end upward. The cross-bar struck the patient across the sternum, just at the manubrio-gladiolar junction. He took two or three steps back, fell to the ground unconscious for a few moments, and was dazed when he got up. He felt a severe burning pain in the midsternal region, with dyspnea and distress in taking a satisfying breath. The burning was also felt in the mid-dorsal region, but did not radiate to the arms, neck or other points of the chest. There was also dizziness after moving.

One month later, he developed inconstant pain in the left nipple region, for which he stayed in bed for one week. He returned to bed at times with a burning sensation in the middle of the chest, and a sticking pain in the nipple region. He became fatigued easily and had faintness at times after the accident. At times, he also felt a sudden rapid palpitation, especially during the night, and slept only spasmodically, waking with pain, burning sensation, dyspnea, and cough. There was no wheezing nor hemoptysis. Gradually, the burning sensation lessened, and he then felt the sensation of a lump in the midsternal region. At time, he also had pain in the same region on deep breathing. For seven months after the accident, he could not lie on either side, and later could not lie on the left side.

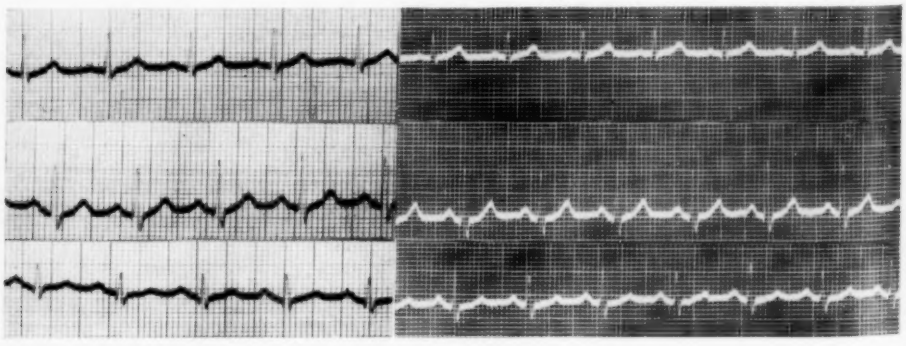
Physical examination a year and a half later showed the heart not enlarged, and the sounds at the apex of good quality. Over the aortic area, there was a soft blowing systolic murmur, but no basic accentuations. There was a tender spot on the third left rib, two inches from the median line, and also tenderness over the sternum just below the manubrio-gladiolar junction. The temporals were tortuous, and the blood pressure was 132/90.

A teleroentgenogram showed the cardiac shadow normal in size and configuration, but the aorta appeared moderately elongated with slight prominence of the descending portion of the arch.

One year later, the patient developed angina pectoris with repeated typical attacks of pain in the precordial and sternal regions, radiating to the left arm. There was weakness and inability to move about with comfort. Examination showed a pulse rate that increased considerably on slight exertion, and a low blood pressure. Otherwise the physical examination showed no new evidences. The electrocardiogram which had previously been negative, at this time showed a conspicuous alternation of the *R* wave. Otherwise, the waves would be considered entirely normal.

The disability was adjudged total and permanent.

Summary—The clinical sequence of events is definite. Following direct injury to the chest wall, the patient became unconscious. The subjective symptoms that followed are the main



(1) Electrocardiogram 1½ years after direct thoracic injury. (2) One year later.

FIG. 5—Christino G.

guide and evidence of some affection behind the sternum. Involvement of the aorta or heart, and even laceration or contusion with ecchymosis may occur following severe injury. Over the aortic area, there was a soft systolic blowing murmur. There was a tender spot on the third rib, two inches from the midline as well as below the manubrio-gladiolar junction. This, together with the x-ray findings, indicates aortic involvement. There is evidence of slight general arterial changes, which are no doubt of the same type as the aortic lesion and entirely independent of the injury. The later development of angina pectoris is corroborative evidence of the localization of the traumatic lesion. The atheromata in the aorta following the injury obviously spread to the coronary arteries and induced the attacks of angina pectoris.

It is mainly, however, by the record of the injury and the patient's symptoms that we are compelled to conclude that there was damage to the aorta with subsequent cardiac symptoms and angina pectoris, all on the basis of the accidental lesion. There may also have been injury to the connective tissue at the base of the heart, in the region of the insertion of the large vessels into their pericardial covering. This is speculative, however. The duration of the symptoms is usually much prolonged. It often takes years before the patient can resume strenuous activity, and the pain often recurs.

The case reported by Joachim and Mays was that of a man aged 25 who had no history, signs or serologic evidence of syphilis, and no valvular lesions, coronary disease, or diffuse myocardial changes.[32] There was evidence of interstitial myocarditis near the site of the aneurysm. The aneurysm was not located at or near the apex. The only discoverable etiologic factor was an injury to the chest at the age of 12 when he was run over by a wagon and sustained a fracture of some ribs on the left side. At the age of 23 he began to have severe attacks of paroxysmal tachycardia with a heart rate of 170-200. These attacks lasted from ten minutes to as long as one week. There was cyanosis, slight enlargement of the liver, and congestion of the bases of the lungs in the severe attacks. He died suddenly. Autopsy showed an aneurysm of the anterior wall of the left ventricle, not near the apex, and composed largely of lime salts. There was also marked interstitial myocarditis and atrophy of the muscle fibres in the surrounding region.

Rupture of the Heart

The relation of trauma to rupture of the heart is well recognized, particularly when it concerns penetrating wounds. Traumatic rupture is generally due either to gunshot wounds or to stab wounds. These will be taken up at the end of this section. We shall here concern ourselves with rupture of the heart due to a non-penetrating injuries. This may follow a blow, a fall, or a compression injury to the chest, as well as violent effort some time after the injury has been received. It was first described, as

[32] Joachim and Mays: Amer. Heart Jour., 1927, ii, 682.

far as we can learn, by William Harvey.[33]

Morgagni, who himself died from it, described it in an old woman of 75, who had the traditional fatty heart.[34]

The symptoms of rupture of the heart are in reality the terminal phenomena that take place. They usually consist of the momentary symptoms of collapse and sudden death.

In the few cases where examination of the heart was made just preceding death, its action was rapid and tumultuous, often irregular, and the sounds of poor quality. A continuous muffled, low-pitched, rushing rumbling has been described as indicating the pouring out of blood into the pericardial sac.[35] The mechanism for the occurrence of death depends upon the intrapericardial pressure.[36] When the pressure in the pericardium becomes equal to that in the right auricle, blood no longer enters the heart from the systemic veins and death promptly ensues.

Traumatic rupture of the heart within an intact pericardium is almost certainly due to compression or concussion. Either of these accidents may directly injure the heart tissue by bruising from without, or else may so raise the intracardiac blood pressure that the healthy heart wall is burst from within. Compression will vary with the amount of colliding force, while concussion will depend on the rapidity of its application. The site of rupture of the heart in traumatic cases is more frequently in the right ventricle than in the left on account of its closer proximity to the anterior chest wall.

Krumbhaar and Crowell report a very interesting case with autopsy findings, showing a very pointed relation of the trauma to rupture of the heart.[37] A man, aged 38, of dissolute habits, during a drunken debauch at Atlantic City, fell heavily against a box for mixing mortar, hitting the edge against his precordial area. When seen several days later, he had considerable precordial pain, but no diagnosis was made. No further details were available except that death occurred suddenly. At autopsy, the pericardial cavity was filled with clots of blood. About half way up the outer wall of the left ventricle in the visceral pericardium, was a transverse tear, 3 cm. in length which extended into the myocardium for about two-thirds of its depth. Histologically, the coronary arterioles were reported as occluded, and the myocardium near the rupture was acutely necrotic.

It is probable in this case that trauma played a prominent part, by injuring the coronary artery with subsequent thrombus formation. Rupture occurred "spontaneously" as a result of the thrombosis.

[33]Harvey, Wm.: Works. Translated by Sydenham Soc. 1847. P. 127.

[34]Morgagni: De Sedibus, Venice, 1761, Epist. 58, No. 14.

[35]Reznikoff: Jour. A. M. A., 1922, lxxviii, 1926.

[36]Hopkins: Proc. N. Y. Path. Soc., 1910-1911 (new series), x, 200.

Francois-Franck: Gaz. Hebdomad. 1877, xiv.

[37]Krumbhaar and Crowell: Amer. Jour. Med. Sci., 1925, clxx, 828.

The following group of cases also present the typical picture of this type of case.

Kennedy reports the case of an emaciated woman of 60 years who was struck in the chest by the shaft of a cart and died in an hour.[38] No external bruising was present. The second, third, fourth, and sixth ribs of the right side were fractured. The pericardium, however, was untorn and healthy, and the heart muscle showed some brown atrophy. In the anterior wall of the left ventricle there were two ruptures on a level with the transverse fracture of the sternum. There were also some patches of hemorrhage under the epicardium, and also hemorrhage in the tissues around the origin of the great vessels. The pericardial sac was filled with blood. There can be little doubt that the blow on the chest was delivered with considerable violence, yet there was no bruising visible externally. The rupture occurred by bursting, due to concussion.

O'Neill records the case of a boy who fell down and was stamped on.[39] He was kept in bed for two days and was then allowed up in the absence of severe symptoms. He was apparently well for four days and died suddenly in bed the following day. Autopsy revealed a hemopericardium and a slit 3 mm. long above the auriculo-ventricular ring.

Turner and Gould report the case of a boatswain, aged 47, who fell forward off his ship, a distance of 20 feet on to a buoy.[40] He was unconscious when picked up, but no external injuries were found. An hour and a half after the accident, he was unconscious, moving his limbs, and shouting in a state of cerebral irritation. He became conscious several hours later, but the heart sounds were distant and feeble, and the radial pulse impalpable. There was no bruising of the chest wall. He died suddenly five hours after the accident. There was a fracture of the first segment of the gladiolus. The periosteum of the deeper surface was torn by the jagged edge of the fracture. The pericardium was filled with blood and there was a small tear in the anterior wall of the right ventricle close to the semilunar valves. There was a small bruise, but no tear of the parietal pericardium immediately in front of this.

Bilderbeck records the case of a young man, aged 19, who was crushed between a stone wall and a wheeled fire-pump as it rolled downhill.[41] The cross bar handle caught him across the middle of the chest. He was seen to fall dead at once. The ribs were unfractured. There was no trace of old disease in the heart muscle or its valves. In the wall of the right auricle there was a small rupture. The pericardium was full of blood, but undamaged. The explanation is that at the moment of impact, the man may have taken a deep inspiration, engorging the right auricle which then burst on sudden compression through the costal wall.

[38] Kennedy: Lancet, 1914, i. 105.

[39] O'Neill: Jour. Am. Assoc., 1914, xii, 697.

[40] Turner and Gould: Lancet, 1917, ii, 567.

[41] Bilderbeck, A. C. L.: Brit. Med. Jour., 1919, i, 675.

Howatt reports a case of traumatic rupture of the heart in an adult male, aged 23. The man fell 45 feet, alighting on a wooden plank and a pile of steel plates.[42] The death was not instantaneous, the patient dying twenty minutes after the accident, in a hospital. Externally there were practically no evidences of injury. Autopsy revealed that the pericardium was distended with blood, due to a rupture of the free edge of the left auricular appendage.

In this case, the injuries were caused by the violent impact of the body against the ground. The nature of the cardiac rupture raises the question as to the mode of its production. Trauma may cause injury of the heart's wall without immediate rupture, but after an interval the injured area may prove the site of a rupture, which may be described as "delayed traumatic" or "spontaneous" according to predilection. Where the interval between the receipt of the injury and death is considerable, difficult questions about the cause of death may be raised by claims for monetary compensation or damages.

Howatt also records a case in a bricklayer aged 68 who was squeezed between a wall and a small passing truck which scraped the skin on the front of his chest, doing no further apparent local injury. He was in bed for two weeks and resumed work after three weeks, in apparent good health, and with normal pulse rate. After three days of work, he felt disabled by pain in the chest and remained abed. His pulse rate rose gradually and after two weeks of rest, he suddenly died. The heart muscle and the pericardium were not the seat of any disease. The left ventricle was bruised in five places, the largest bruise, in front near the apex, extending through the greater part of the wall's thickness. Here the ventricular wall was ruptured and the pericardial sac was filled with blood.

Howatt also reports three other cases of traumatic rupture with intact pericardium. One resulted from a shell wound of the skin in the left mid-axillary line with comminuted fracture of the fourth and fifth ribs. The anterior wall of the left ventricle showed a vertical perforation. The pericardium was intact. Such an injury points to contusion of the heart wall by the broken ribs acting through the pericardium, and concussion from the violence of the trauma.

In another gun-shot case, there was a non-penetrating wound of the thoracic wall, without fracture. The anterior wall of the right ventricle showed a minute rupture and the pericardium was distended with blood.

Spontaneous rupture of the heart may bear a definite, although somewhat delayed relationship to direct injury to the chest. This is illustrated by the case of Krumbhaar and Crowell above recorded, and the following three cases in which death took place at some time after injury, where no immediate effort or trauma seemed to play any part. Two are by Dreack, one, a workman of 58, who nine months before his sudden death had been struck twice on the chest and

[42]Howatt, R. K.: Lancet, London, 1920, i, 1313.

visibly bruised.[43] Subsequently he continually complained of pain in the chest. Fourteen days before death, he developed influenza and at autopsy was found to have a rupture of the apex in an aneurysm which had developed subsequent to the injury. It was therefore considered that it was a "spontaneous" rupture with trauma as the underlying cause.

The second was a girl of nine who had fallen down stairs five weeks before death, which occurred suddenly on getting out of bed. The rupture in the right ventricle was thought to be dependent on the former trauma. The muscle was said to be normal. The third case is reported by Corin and concerns a miner of 62 who, fourteen days before his sudden death, had been crushed by a wagonload of coal, but did not stop work at the time.[44] There was no external lesion or bruise of the thoracic muscles or bones at autopsy, and the author thought that the rupture of the left ventricle should be traceable to the history of traumatic injury to the heart.

In the etiology of those cases in which spontaneous rupture takes place following exertion, coronary sclerosis holds a pre-eminent place. In consequence, there develops ischemic necrosis with thinning and replacement fibrosis, and the formation of aneurysm of the left ventricle due to the high intraventricular pressure. Fatty infiltration of the heart muscle or brown atrophy due to the slow coronary sclerosis of age may exist. In all these cases, the sudden increase of blood pressure during work plays a critical role in precipitating the rupture of the heart.

From experiments on animals it seems that on suddenly produced insufficiency of the aortic valve, the intraventricular pressure may become so great as even to rupture the unprepared ventricle. In one such case reported, there occurred also sudden rupture of the posterior pillar of the lower mitral cusp.[45] It is almost certain, as was maintained by Morgagni, that a complete rupture never occurs through the sound heart muscle.

Penetrating Wounds

Besides rupture and laceration of the organ produced by contusions and crushing injuries, external penetrating wounds of the heart occur, passing through one or more of the heart walls. Gunshot wounds constitute a large percentage of the penetrating wounds of the chest. In addition to these, fractures of the ribs and sternum play an important role. In these cases, the sharp end of a broken fragment may directly lacerate the tissues of the heart. The seriousness of these injuries depends upon the amount of trauma inflicted upon the intrathoracic structures.[46]

Penetrating wounds of the thorax

[43]Dreack: Giessen Dissertation, 1909. This and a number of other references to the subject were kindly supplied by Dr. E. B. Krumbhaar of Philadelphia, to whom we are deeply grateful.

[44]Corin, M. G.: Bull. de l'Acad. Roy. de Med. de Belg. 1911, xv, 563.

[45]Bouillan, quoted by Allbutt, System of Medicine, 1909, Vol. vi, p. 425. MacMillan & Co., London.

[46]Keen: Surgery. Saunders, Philadelphia, 1916. Vol. v.

associated with wounds of the pericardium or heart are always accompanied by severe shock. When the great vessels are involved, death generally follows in a few moments. These conditions must always be considered serious and naturally treated as emergencies.

The usual site of the penetration is one or the other of the right chambers of the heart, as the wound is generally received from in front. The wound in the pericardium permits of escape of blood, and thus lessens the risk of fatal embarrassment of the heart's action by the increased intrapericardial pressure due to hemopericardium.

The prognosis in penetrating wounds of the chest is always serious. Yet, a number of cases have been amenable to immediate repair with subsequent recovery. In these cases, certain posttraumatic disturbances of the heart may result. Adhesive pericarditis may develop after recovery. If there is lodgement of the foreign body in the myocardium, removal will result in a localized lesion which may be the focus for extrasystoles.

Freese reports a case of a negro who was stabbed and immediately operated on while he was unconscious, with feeble respirations, imperceptible pulse, widely dilated pupils, and cold perspiration.[47] There was a wound half an inch long, in the fourth space to the left of the sternum. After removal of part of the fourth and fifth left ribs and the costal cartilages, a wound of the pericardium was seen exuding blood. When the clot was removed from the pericardial sac, the heart wound was seen in the posterior wall of the left ventricle, a little above the apex. This was sutured, the pericardial wound closed, and the musculocutaneous flaps replaced on the chest wall. The patient recovered without any subsequent cardiac embarrassment.

Under this heading belongs also the consideration of the question of injuries to the blood vessels and thoracic wall. These frequently complicate penetrating wounds. The internal mammary artery and intercostal arteries may give dangerous and even fatal hemorrhages within the chest. The rupture of the larger vessels in the thorax such as the superior vena cava has also been discovered following penetrating wounds.

In one case, reported by Stephens, a man of 42 fell into a cellar, fracturing the sternum at the sternal angle.[48] On opening the chest, a marked extravasation of blood was found behind the site of the fracture, due to a tear in the superior vena cava.

There is a case reported by Fisher of a negro 24 years old who was stabbed just within the left nipple.[49] He was in extreme shock with cold skin and barely palpable pulse. Under ether anesthesia, blood was found to escape from a wound in the pericardium and an attack of paroxysmal ventricular tachycardia was observed. There was also a penetrating wound

[47] FREESE: Jour. A. M. A., 1921, lxxvi, 520.

[48] STEPHENS: Lancet, London. 1922, ii, 1382.

[49] FISHER, J. L.: The Repaired Heart. Jour. A.M.A., 1926, lxxxvi, 192.

over the left ventricle, one inch long which was grasped and sutured. The patient recovered.

There are recorded in the literature several such interesting cases. Some of them are of stab wounds which required suture and resulted in complete recovery.

Compensation Criteria

In considering the subject of trauma to the thorax with intrathoracic cardiovascular injury from the point of view of the compensability of the accident, several important criteria must be established:

1. From a labor standpoint, the heart is healthy if a man is able for a long period of time to pursue his occupation without distress and without long periods of absence from work.

2. If, following direct or indirect violence to the chest, signs of an intrathoracic cardiovascular lesion develop, which are incapacitating, they must be considered the result of an aggravation of a previously-existing asymptomatic lesion, or the result of damage to a previously normal heart.

3. As in heart strain, the time that elapses between the accident and the development of disabling symptoms is very short. There must be immediate pain with its concomitants—dyspnea, rapid irregular pulse, faintness, and cold sweat, and immediate partial or total disability, in order that causal or aggravating relationship be clearly established. Temporary improvement with return to usual or lighter work, followed by a recurrence of the condition may occur. But in these cases, the re-appearance of the symptoms and signs should be attributed to the original injury.

Summary

1. The superficial position of the heart and pericardium directly behind the sternum and the adjacent cartilages exposes it to danger from injuries to the anterior chest wall.

2. With or without injury to the ribs, or even without obvious external bruising, an external blow may produce very serious damage to the intrathoracic structures.

3. The principal clinical types of traumatic heart lesions that occur in consequence of direct and indirect violence are classified.

4. The pericardium may react to a contusion or concussion of the chest with the development of acute pericarditis.

5. Auricular fibrillation may follow direct violence to the chest. Illustrative cases are reported by the authors.

6. Extrasystolic arrhythmia may arise following damage produced in the cardiac tissue by direct injury to the organ from blunt violence such as blows to the chest, or indirect injury as in falling from a height.

7. The location of the trauma in the myocardial structure will predicate the functional disturbances or arrhythmia that may develop. Cases are reported of heart block from direct injury to the chest.

8. Injury to the front of the chest, with or without injury to the ribs or even without obvious external bruising, may cause rupture of one or more of the valves of the heart.

9. The mechanism of the production of such injuries is discussed.

10. The most important clinical characteristic of these cases is the immediate development of physical signs referable to the valvular lesion.

11. Besides injury to the aortic cusp, the wall of the vessel frequently suffers in accidents due to direct violence to the front of the chest, with the formation of a dissecting aneurysm.

12. A pre-existing aneurysm may rupture in consequence of direct blunt violence to the thorax.

13. A number of cases in the literature indicate strikingly that due directly to external non-penetrating violence, small foci of myocardial damage may develop.

14. It must be recognized that milder injuries of the myocardium may take place consistent with life over a varying length of time.

15. Rupture of the heart may follow non-penetrating injuries to the chest, as well as violent effort, some time after the injury has been received.

16. The symptoms of rupture of the heart are in reality the terminal phenomena that take place. They usually consist of the momentary symptoms of collapse and sudden death.

17. The mechanism for the occurrence of death depends upon the intrapericardial pressure. When the pressure in the pericardium becomes equal to that in the right auricle, blood no longer enters the heart from the systemic veins and death promptly ensues.

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On Measurements of the Renal Veins: A Contribution to the Literature of Postural Albuminuria

By Clarence Quinan, M.D., *San Francisco, California*

IT may be affirmed as literally true that our knowledge concerning the mode of origin of the so-called physiological albuminurias is not much greater now than it was forty years ago. Indeed in the somewhat extensive literature that deals with these functional disorders there is only one necropsy report to be found and according to that report (1) the examination was negative in its results except that a small and apparently insignificant lesion was found in one kidney. It would therefore seem justifiable to bring forward objective data of almost any description, if only they enable us to make progress toward the discovery of a causative mechanism. In pursuance of this idea, I describe here very briefly various clinical and other data noted down from year to year during the study of what seems to have been a remarkable, if not a unique, case of postural albuminuria. The patient, a young man who had had a severe form of this disorder since childhood, finally succumbed to an attack of broncho-pneumonia. The renal vein measurements recorded in this paper were made in consequence of certain unusual facts observed during the post-mortem examination.

As I have already published an account of this patient's clinical history, (2) for the present purpose it will suffice to outline the main features of his case.

Report of Case

History—T. T., a man, aged 23, seen first, Jan. 20, 1915, who appeared to be in good health and whose family history was unimportant, stated that in his seventh year, following an attack of whooping cough, albumin was discovered in his urine. It had persisted ever since. Apparently, however, the trouble had not affected his health very materially, since for a number of years he had led an active out-of-doors life as a "field-naturalist."

Examination.—The patient was well developed, muscular, and his figure was erect, athletic and showed no trace of lordosis. His height was 5 feet 8 inches (174 cm.), and he weighed 148 pounds (67 kg.). With the exception of a slight facial pallor, he looked physically fit. The right kidney was freely movable; the left could not be felt. In other respects, the examination of his trunk and extremities was negative in its results. The eye grounds were normal.

The urine contained a large amount of albumin, and a few hyaline casts were found in the sediment. He was instructed to bring for examination samples of his urine collected (1) before rising from bed in the morning, and (2) after being up and about for some time. These specimens, and another voided at the office, contained, respectively, 0.75, 6 and 7.5 parts per thousand of albumin.

It was not until May 4, 1922, seven years later, that I was enabled to resume the study of this patient's case.

His life during this intervening period had been an extremely active one. For he had served throughout the war as an officer in the aviation corps of the A. E. F.—he said that he had been enabled to pass the physical examination required in this branch of the military service simply by substituting for his own urine that of a comrade; and, since the war, he had been an outdoor solicitor for a steamship company, a line of work which had kept him almost constantly on his feet.

May 4, 1922, his general physical status remained much the same as it had been in January, 1915. His blood pressure was 122 systolic, 85 diastolic, and it was not appreciably modified by change of posture. The eye grounds were normal. The albuminuria persisted.

As it seemed possible that the massive output of albumin characteristic of all urine specimens voided by this patient when he stood erect might be related in some way to the fact that his right kidney was freely movable, numerous experiments were made with a specially devised apparatus by means of which the kidneys, either singly or both together, could be quite securely held in place. Without exception, however, these experiments were negative in their results; for quantitative tests made with specimens of urine collected by this patient while upright, with the apparatus in place, showed that the albumin output was not in the least influenced by fixation of either one or both of the kidneys. On the other hand, it was proved by experiments that his clinostatic albuminuria could be notably reduced in amount through the simple expedient of having him sleep in a bed the foot of which had been raised about six inches.

Although the urine of this patient was heavily albuminous at all times, it was characteristic that the maximum output of albumin in the urine occurred only after he had left his bed and had been up and about for some time. The average rate of albumin elimination was determined by Esbach's method. The tests—120 in all—were made at regular intervals during a period of five months. The average values thus obtained were 1.24 parts per thousand for the specimens collected in the horizontal posture, and 4.30 and 7.60, respectively, for the specimens voided while he was on his feet at 7:20 and at 7:40 A.M.—see Fig. 1.

That the postural albuminuria was remarkably constant in character can be seen at a glance by comparing the data noted at different times during a period of eight years, see Table 1.

Table 1—Albumin Elimination During a Period of Eight Years in a Case of Postural Albuminuria

	Albumin in Parts per Thousand		
	7:00 A.M. Patient Horizontal No. 1	7:20 A.M. Patient Vertical No. 2	7:40 A.M. Patient Vertical No. 3
Jan. 20, 1915	0.75	....	6.0
May 4, 1922	2.25	4.00	8.0
Jan. 18, 1923	0.90	3.75	5.0

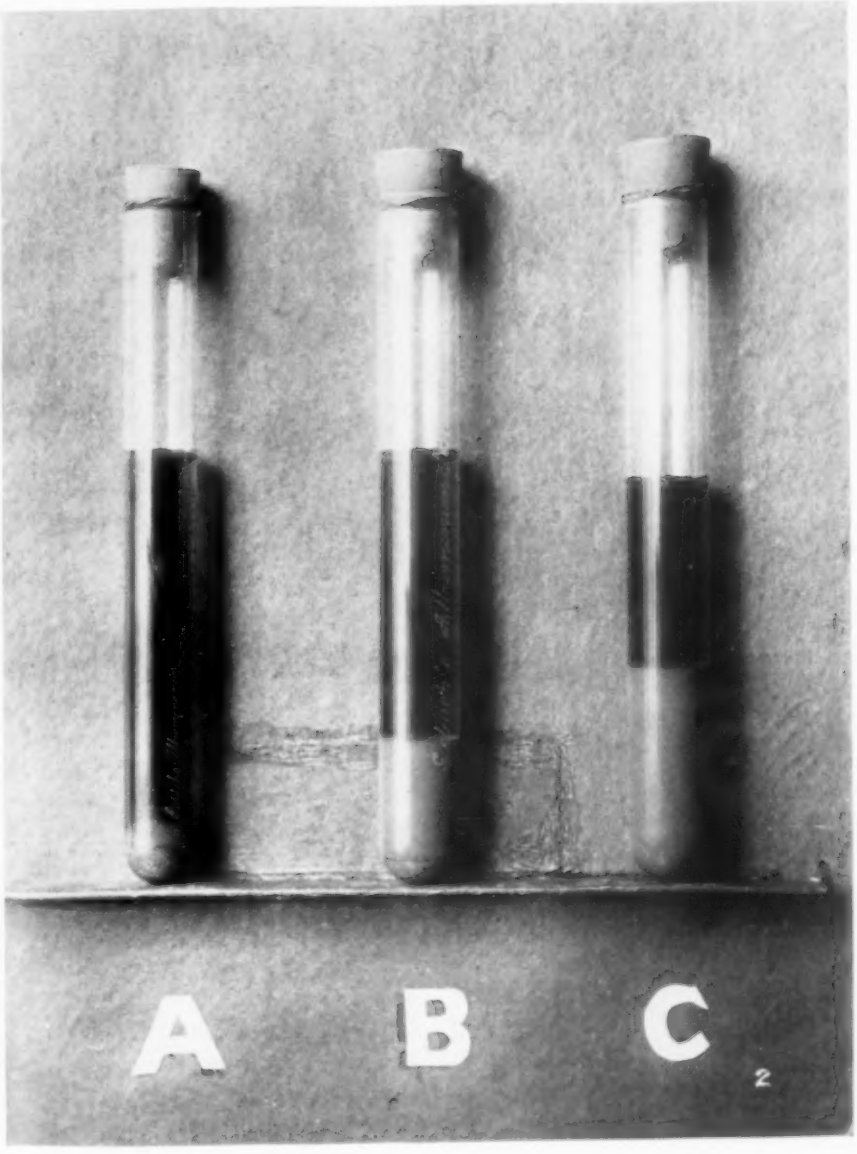

FIG. 1. Typical Esbach tests: Appearance after twenty-four hours: A, horizontal specimen, 7 A.M., patient recumbent; B, first vertical specimen, 7:20 A.M.; second vertical specimen, 7:40 A.M.

January 28, 1923, in consequence of having been chilled through exposure, he contracted broncho-pneumonia and died after an illness of eight days.

The post-mortem examination showed that death had been due to broncho-pneumonia, complicated with acute bilateral pleurisy and acute suppurative pericarditis; but it also disclosed an extremely remarkable condition of visceroptosis, as may be seen from the following report as to the abdominal findings.

Autopsy Report.—The liver projects over a hand's breadth on both sides. The stomach is pushed far down. The fundus is three fingers below the umbilicus. The transverse colon is a little above the symphysis. Most of the small intestine is in the pelvis.

The right kidney is rather loose, and it is distinctly displaced downward. The lower edge of this kidney is just opposite the crest of the iliac bone, on top of the ileopsoas muscle. The hepatic flexure is fastened in this same region. The transverse colon then runs in a large U-shaped loop down to the symphysis and up along the greater curvature of the stomach to the lower pole of the left kidney which is in its normal position, slightly looser than it should be.

The liver is displaced downward and it is somewhat loose in its attachments posteriorly. It is unusually large, easily one and a third times its normal size. Both lobes are equally enlarged. Its shape is normal. The surface shows passive congestion and edema. The hepatic artery is normal. The liver weighs four pounds (2154 grams).

The two renal veins are both very wide. They arise from the vena cava at the usual level. The left renal vein measures 18 millimeters, the right 16 millimeters in diameter.

The left kidney capsule strips easily. The kidney is smooth and purplish in color. It measures 125 by 55 by 25 millimeters. Small, irregular, ill-defined yellow spots are visible on the surface. The left kidney weighs 170 grams.

Cut Surface: The cortex measures approximately six millimeters. It is rather opaque and slightly yellowish. The columns of Bertini are of the same color. The pyramids show slight passive congestion; otherwise they are normal.

The right kidney capsule strips easily. It is slightly thickened and more whitish than the left. The cut surface is smooth. This kidney measures 102 by 60 by 28 millimeters. It weighs 113 grams.

Histological Examination.—(a) Liver: The sections show moderate peripheral fatty degeneration. The arteries are normal.

(b) Kidneys: There is fibrosis of many glomeruli, much new fibrous tissue with atrophy of tubules and moderate round-celled infiltration; here and there comparatively normal tubules show much granular and some fatty degeneration. Some of the cells seem to be full of crystals of fatty acid; few casts; slight endarteritis of some of the arteries. In places the new connective tissue appears to contain large, irregular granules of calcareous material.

Measurements of Renal Veins

In view of the fact that a marked postural albuminuria had been for many years this patient's only symptom, it was natural to be struck by the circumstance, noted in the autopsy report, that both of his renal veins appeared to be very large; but, of course, no significance could be attached to such a finding unless it could be shown that the vessels mentioned were, in fact, oversize. Since, however, the text-books are virtually silent on the subject of the renal veins, and say nothing as to their variations in caliber, in order to determine the average size of these vessels it became necessary to attack this problem at first hand. Accordingly, with the end in view to obtain such values, I have

measured the collapsed, empty renal veins in a number of human subjects. The data accumulated in the course of this work are shown in Table 2.

TABLE 2—RENAL VEIN MEASUREMENTS IN 100 UNSELECTED SUBJECTS

P-M No.	Age and Sex	Widths of Veins In mms. R	L	P-M No.	Age and Sex	Widths of Veins In mms. R	L	P-M No.	Age and Sex	Widths of Veins In mms. R	L	P-M No.	Age and Sex	Widths of Veins In mms. R	L
1	M60	11	10	26	M25	11	12	51	M42	12	12	76	F 39	12	10
2	M60	10	11	27	M42	11	10	52	M40	9	10	77	F 45	11	12
3	M58	12	10	28	F 31	13	11	53	F 68	11	12	78	F 50	9	14
4	F 35	11	12	29	M28	11	11	54	M60	9	10	79	F 70	11	14
5	F 25	14	11	30	M45	8	13	55	M60	10	12	80	M60	14	12
6	M50	12	13	31	M60	10	11	56	M55	11	13	81	M50	10	12
7	M35	10	11	32	M60	10	14	57	M35	11	10	82	M75	13	10
8	M34	9	9	33	M45	8	14	58	M41	11	10	83	M40	12	10
9	M45	8	14	34	F 71	11	9	59	M39	10	15	84	M45	8	11
10	M52	11	13	35	F 54	9	10	60	M72	12	11	85	M29	9	12
11	F 35	9	10	36	M40	11	7	61	M36	9	11	86	M38	11	12
12	M25	12	7	37	M27	14	13	62	M40	11	12	87	M60	10	10
13	M50	11	11	38	M75	9	11	63	F 42	10	13	88	F 29	14	12
14	M30	14	13	39	M55	14	13	64	M40	11	13	89	M55	10	11
15	M70	15	11	40	M40	9	11	65	M34	12	16	90	F 14	10	11
16	M45	11	10	41	M19	10	10	66	F 26	10	14	91	M35	12	13
17	F 45	10	11	42	F 30	13	11	67	M32	8	10	92	M40	9	11
18	M22	10	11	43	F 28	10	13	68	M45	10	10	93	F 25	13	11
19	M72	10	12	44	M50	12	11	69	M55	10	10	94	M25	12	14
20	F 45	10	12	45	M70	12	9	70	M45	11	12	95	F 42	12	13
21	F 82	9	8	46	M60	9	15	71	F 40	10	12	96	M45	10	13
22	M40	11	13	47	F 60	12	11	72	F 21	10	11	97	M45	7	11
23	M55	12	10	48	M17	9	13	73	M50	12	12	98	M60	12	11
24	M36	13	12	49	M40	16	10	74	M60	9	10	99	M45	9	13
25	F 60	13	11	50	M55	10	14	75	F 34	11	10	100	F 40	8	11

Mean values in mms.: Right vein 10.7, left vein 11.4.

The subjects examined were unselected, except that those only were turned to account whose renal veins were represented by the usual single, straight trunks. As the work reported here was done largely in the San Francisco city morgue, it will be understood that, from the standpoint of pathology, the material employed was quite diversified in character; but it should be stated that cardio-renal disease was perhaps the most common cause of death, and that not a few of the subjects were those of apparently normal individuals who had met death through some form of violence.

In so far as the main objective point of this research is concerned, it will be noted (see Table 2) that the average values obtained for the widths of the renal veins are 10.7 millimeters for the right vein and 11.4 millimeters for the left. This gives 22.1 milli-

meters as the average total cross-section of these veins.

However, besides yielding these average values, the data brought together in this paper, though comparatively few in number, also justify three other seemingly definite conclusions. These are: 1. That there is no fixed rule as to the relative widths of the two renal veins. In the present series, for example, the left vein was larger than the right in 57 subjects, in 34 subjects the right vein was larger than the left, while in 9 both veins were of the same caliber. 2. That the total venous path of the kidneys, as shown by the combined diameters of the renal veins, varies within comparatively narrow limits. In a majority of the subjects that I examined it ranged from 19 to 26 millimeters; and since in but a single instance was a value of 28 millimeters noted, it seems probable that values in excess of this figure are probably to be regarded as exceptional. 3. That the total venous path sustains no relation to the total mass of the two kidneys. As a matter of fact, having made careful measurements of each of the two-hundred kidneys used in this series, I can state that as a rule the largest veins occurred in kidneys of medium size whereas, not infrequently, kidneys of relatively huge dimensions had very small veins.

Now, according to the autopsy report, the right and left renal veins of the patient, T. T., measured, respectively, 16 and 18 millimeters. In this instance therefore the blood vessels mentioned had a total cross-section of 34 millimeters. However, on comparing these values with those recorded in Table 2, it becomes evident, not only that each one of the renal veins was unusually large, but that the figure which represents their combined diameters exceeds by more than fifty per cent the corresponding average value obtained in the present study of 100 unselected subjects.

Comment

Here, then, we have to do with the case of a young man of superb physique, who undoubtedly suffered from chronic nephritis, but whose major and almost only symptom had been for many years a massive albuminuria always exaggerated by the change from the horizontal to the vertical posture. As to the cause of this the main presenting symptom, needless to say, it is only possible to indulge in conjectures that seem to be in keeping with observed facts. Having in mind, however, that the post-mortem examination disclosed a huge liver, loose in its posterior attachments, together with a general downward displacement of other abdominal organs—to say nothing of the greatly enlarged renal veins—I am disposed to believe that in this particular instance both the postural albuminuria and the widening of these veins may have been the end of results of a constantly recurring passive congestion. This, I believe, may have been caused by a partial constriction of the vena cava, probably at the point where it penetrates the diaphragm—that is to say, at the foramen quadratum. As to the nature of the constricting force, I venture to affirm that it may have arisen through a traction-tug exerted upon this great blood vessel by the pro-

lapsed abdominal organs. In other words, I believe that the postural albuminuria was indirectly due to the visceroptosis.

Conclusions

1. The average values obtained for the widths of the renal veins, as determined by measurements of these blood vessels in 100 unselected human subjects, were 10.7 millimeters for the right vein and 11.4 millimeters for the left.

2. The average total cross-section value for these vessels was found to be 22.1 millimeters.

3. Exceptionally, one or other of the renal veins may reach a diameter of 16 millimeters; but it is rare for the combined diameters of these vessels to measure over 27 millimeters.

4. Details are given as to the clinical history and the post-mortem findings in a remarkable case of postural albuminuria.

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Massive Atelectasis*

Clinic of C. H. Cocke, M.D., F.A.C.P., *Asheville, North Carolina*

THE subject for discussion this afternoon is a very brief resumé of so-called massive collapse of the lungs with the addition of case reports of two interesting unpublished cases.

"Massive Collapse" was first described by the Englishman, W. Pasteur, in the year 1890 after study of 7 autopsy cases which presented post diphtheritic paralysis of the diaphragm, the result of nasal and pharyngeal diphtheria. It was not observed in laryngeal cases. The same author twenty years later, in 1910, described "post operative massive collapse" in a study of 2,000 operations in which there was an incidence of 201 post operative pulmonary complications, of which pulmonary complications, 16 or 8% were in the nature of massive collapse. These operations were mostly abdominal sections, chiefly for appendicitis, and herniotomies. No case was observed following operations on the head and upper extremities.

Sir John Rose-Bradford — 1918-1920—discovered in a study of war wounds of the chest, whether penetrating or non-penetrating, that 5% to 10% of these were succeeded by the development of more or less massive collapse, but found no evidence of bronchial obstruction, pleural effusion, or any other lesion interfering with the aeration of the lung.

Scrimger in 1921 was the first American to report on the condition, recording seven cases. Within the past seven years the literature on the subject, particularly of that form known as post operative massive collapse, has grown quite rapidly, although conclusive evidence as to the true cause of the condition has never been developed.

It is unfortunate that the early writers have given it the name "Massive Collapse" for it is in no sense of the word a true collapse. The condition is one of airlessness of the lung—atelectasis — usually developing with spectacular acuteness, particularly in the post operative and chest wound cases, though developing not infrequently in a more chronic manner as I shall show in my second case.

Quite an interesting and puzzling feature of the cases following thoracic wounds (and it may follow with such dramatic suddenness that the possibility of infection being the etiological factor can be ruled out by the absence of demonstrable infection and the very suddenness of its onset) is that the condition may be homolateral, contralateral, or bilateral. Nor is the condition always massive but not

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infrequently may be only lobar or even lobular in extent. That no true collapse of the lung exists is well shown by the fact that there is present a markedly accentuated *negative* intrapleural pressure (too high to be recorded by the ordinary water or mercury manometer of the usual artificial pneumothorax apparatus) in contradistinction to the positive pressure accompanying a true collapse. Nor has it been conclusively proven that bronchial obstruction is present though the most recent studies of W. E. Lee and Chevalier Jackson, would suggest such a possibility.

The picture of the acutely developed case is briefly: a patient who (say, for illustration, has from one to four days previously had an abdominal operation) has apparently a normal set of lungs and bronchi, suddenly without evidence of infection, respiratory or otherwise, and without other demonstrable evidence of serious or impending complications in the midst of a normal post operative convalescence becomes acutely ill with some form of rather malignant pulmonary mischief. Pain may be present or absent, but there is quite marked cyanosis without evidence of previous cardiac impairment, though tachycardia is common and prominent. Dyspnea is usually quite a definite feature, depending for its extent largely upon the degree of the atelectasis. A cough develops which is at first non-productive though later there is mucoid, and still later muco-purulent sputum—never the bloody or prune juice expectoration of pneumonia—an important point in the differential diagnosis. Chills have occasionally been reported, the rule being for the development of temperature up to say 102 F. or 103 F. The white blood cell count usually runs up to about 20,000. The patient is seen to be suddenly acutely ill, and uncomfortable, though it is not always that the attention is immediately drawn to the respiratory tract.

In the well developed case, the physical findings show a cyanotic, dyspneic patient with marked immobility of the affected side of the chest and narrowing of the interspaces on this side. Vocal fremitus, at first increased, as the condition develops, becomes diminished and then absent with the complete airlessness of the lung. There is dullness to flatness over the affected area, simulating massive fluid, possibly hyperresonance over the rest of the chest, and the further absence of all true vesicular breath sounds (only tubular or transmitted tracheal breathing being heard) may cause a similar confusion. Complete absence of adventitious sounds is found and is most important. There is a high immobile diaphragm and the most characteristic finding of all is the retraction of the heart and quite frequently the trachea to the affected side. This, of course, is in marked contrast to fluid which naturally pushes the heart and mediastinum away from it. The fluoroscope and stereoscopic films confirm these findings and show a homogeneous density coextensive with the atelectasis. Such a picture as the above is for all practical purposes, pathognomonic—though until the condition is completely developed, you may well be confused by the rather bizarre (because not too common) sequence of events and development. You can

quite readily see then, that you must differentiate the condition from pneumonia, acute dilatation of the heart, pulmonary embolus and infarct, pleurisy, with and without effusion, pneumothorax, and diaphragmatic hernia.

Incidence

The condition may occur—

(1) Congenitally

(2) Spontaneously in the course of diaphragmatic pleurisy—and in the new born without demonstrable defect or disease, as I shall show in my first case.

(3) In infections of the lungs and bronchi, such as pneumonia, purulent bronchitis, and tuberculosis (usually chronic).

(4) With post-diphtheritic diaphragmatic paralysis (Pasteur).

(5) Post-operatively and following chest wounds, penetrating and non-penetrating.

(6) Due to bronchial obstruction by tuberculosis, neoplasms (my second case) foreign bodies, mucous secretions, etc.

Unfortunately time forbids a discussion of the interesting speculation as to its cause.

The First Case

(Service Dr. J. LaB Ward, Biltmore Hospital)

M. E. H. Female infant, born March 6, 1927. Normal delivery and child appeared normal in all respects. No anomalies or birth injuries.

Chest—Equal expansion on both sides and breath sounds good. Apparently a perfectly normal baby until the beginning of the fourth day at which time she became acutely ill. There was rise of temperature, rapid pulse, dyspnea, cyanosis, temperature to 102 F. with the entire left chest, anteriorly and posteriorly, flat to percussion with very distant breathing and no râles heard. A little patch, thought to be a pleuritic rub, to the left of the nipple. Right lung-hyperresonant.

Blood—Hbg—95%. R.B.C.—4,800,000. W. B.C. 22,500. Differential—Neut.—56%. S. L.—29%. L. M. 15%.

X-Ray—Pictures made as shown (Fig. 1) and because of the insistence of one of the consultants and the roentgenologist that fluid was present an exploratory tapping was attempted with negative results. Neither the pediatrician nor roentgenologist who insisted on exploratory needling, however, was able to offer any convincing theory as to how such massive fluid could possibly occur in a four day old child, born healthy and possessing an apparently normal heart and lungs.

Temperature range was from 99 F. to 101 F. for three days and then the child completely recovered as shown in the films (Fig. 2) made six days later than the onset of the acute illness, with complete clearing of the physical signs in the chest.

This undoubtedly is a clear cut case of massive atelectasis of the left lung in an infant four days old who was born perfectly normal and has a family history of no bearing whatever on the case. The most probable explanation is that the main bronchus was blocked by a plug of mucus which was coughed up and the lung immediately became aerated again.

Second Case

W. T. C.—Male—Commercial Agt. R. R. Office work. 42-M. Wife and three children living and well. One office mate left 2 mos. previous to Mr. C's illness on account of tuberculosis. Otherwise no contact with tuberculosis.

Family—Childhood and past history of no significance.

FIGURE 1. *M. E. H.*, March 10, 1927. Complete homogenous opacity of left lung.

Habits—Heavy cigarette (30 a day) smoker for 25 years.

P. I.—Patient felt perfectly well until June 4, 1927, when he developed a dry cough. Physical examination; X-ray examination—(Fig. 3) all negative. Five weeks later, cough persisting (July 10, 1927) he had a small pulmonary hemorrhage. Went to bed but three weeks later had two small hemorrhages, then having P. M. temperature to 99 4/5 F. Some cough and expectoration, though little pus in it. Dyspnea only on exertion. Patient kept in bed another three or four weeks and then sent to Asheville, Sept. 29, 1927, with suspected tuberculosis. Patient was spitting a slightly tinged sputum which rapidly cleared, but ran temperature from 98 F. to 100 F. daily with only slight, if any, amelioration of cough. Eighteen days later patient was strong enough to come to office for examination at which time the essential details of the examination were as follows:

Physical Examination — Tall, slender, poorly nourished man of 42. Ht. 70". Wt. 123 lbs., pale, but not cachectic, anxious and worried. Fingers show some clubbing with considerable curving and linear ridging of the nails.

Chest—Long, narrow, thin, with right shoulder droop and marked limitation of expansion of the right side. Vocal fremitus and resonance increased over rt. upper lobe, diminished below on this side—absent over lower one-half.

Heart—Apex in IV interspace 3" to right of mid sternum. Action—rapid, regular, no murmurs. Blood pressure—90-70. Percussion showed dullness over rt. upper lobe

FIGURE 2. *M. E. H.*, March 16, 1927. Complete clearing of left lung.

soon merging into flatness from 3rd rib down—anteriorly and posteriorly—left being clear.

Auscultation — Showed tracheal breath sounds over rt. upper lobe—absent breath sounds elsewhere on rt.—exaggerated inspiration over entire left lung—no adventitious sounds anywhere.

Films—(Fig. 4.)

Urinalysis and Sputum Exams.—Negative.

Blood—Hbg. 70%. R.B.C. 3,840,000. W. B.C. 9,140. Differential—uninforming. Wassermann—negative.

You will note in the film of July 12th (Fig. 3) that we have an essentially normal chest picture. The roentgenologist, Doctor John H. Edmondson, who made the films reported as follows:

"*Chest: Rt. Side:* Some increased fibrosis is noted in the hilum, which contains a number of calcifications in its upper portion.

Left Side: Hilus, bronchioles and parenchyma present nothing from X-ray standpoint suggestive of pathology.

Heart—Centrally placed and about normal in size.

There is *no* impairment to diaphragmatic excursion. This case does not present the appearance of a tuberculous manifestation." When sent to

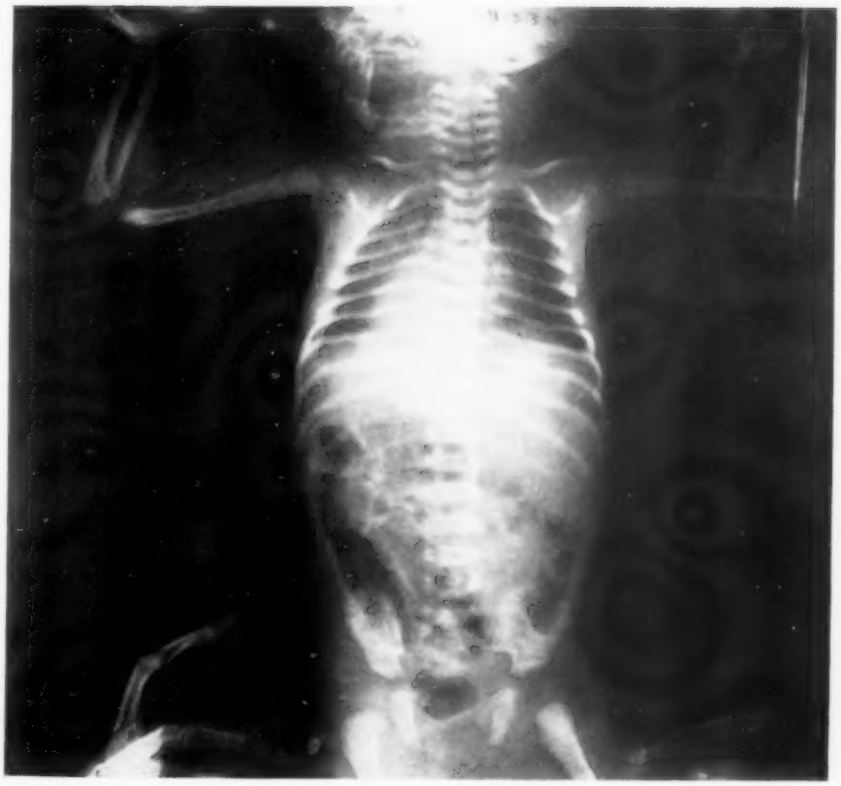

FIGURE 3. *W. T. C.*, July 11, 1927. Apparently normal chest with heart in correct position.

Asheville it was hoped that the patient was suffering from some form of "atypical tuberculous pleurisy" though the transmitting physician had had no opportunity for thorough examination of the patient owing to the continuing hemoptysis. Here is a patient then of 42, an inverterate smoker of cigarettes for 25 yrs.; 46 lbs. underweight, though only 10 lbs. to 15 lbs. under his personal average; presenting the history of a harassing cough developing without a coincident respiratory infection, this cough at first dry with later some muco-purulent expectoration, and repeated hemoptysis with daily temperature to 99° or 100 F. As you note, X-ray films made prior to any blood spitting, but because of suspected tuberculosis, revealed an essentially normal picture, with the heart in its proper position. Some ten weeks after the hemoptysis when I saw the patient, the entire picture was changed but it was not at first apparent what was the true condition. For two weeks after arrival patient was not subjected to thorough chest examination for fear of causing return of the hemoptysis. The blood picture had immediately cast suspicion upon the probability of this

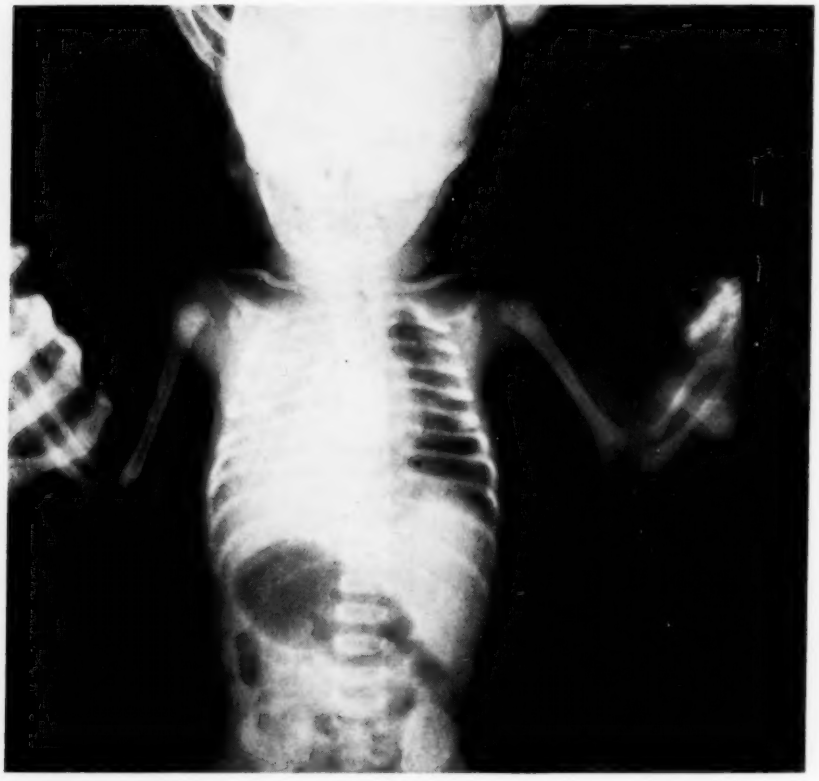

FIGURE 4. *W. T. C.*, Oct. 18, 1927. Complete opacity of right chest with heart and trachea pulled to right side of thorax.

being a case of pulmonary tuberculosis. Such a degree of anemia as 3,800,000 R. B. C. in a patient who showed no demonstrable tuberculous pathology only three months previously and who had not lost, all told, in two months over a pint of blood did not lend weight to the possibility of tuberculosis.

The patient was brought to the office because of a suspicion that a more serious state of affairs was going on than tuberculosis. The clubbing of the fingers seemed to be increasing, the temperature showed no change under bed rest on a sleeping porch, he had been told that it was not proven that he had tuberculosis, that all sputum tests had proven negative and the risk of further hemorrhage was thought to be small. The films showed, as you see, a complete dextrocardia (of a heart 3 mos. previously in a normal position), a trachea pulled well to the right, and uniform massive density of the entire right chest. With the physical findings as outlined earlier, it was now apparent that we were dealing with a case of complete atelectasis of the right lung. The cause of this it was difficult to ascertain. Pulmonary hemorrhage from

tuberculosis with organization of a clot resulting in plugging the main stem bronchus did not seem likely—particularly as there was no evidence of tuberculosis. The general appearance of the patient, the rapid clubbing of the fingers, especially the anemia out of proportion to probable tuberculosis, the persisting P. M. temperature daily, and the failure of the patient to improve caused the suspicion of malignancy. A tentative diagnosis then of endobronchial malignancy was made which seemed to explain the whole history from harrassing dry cough, with subsequent hemoptysis to the development of massive atelectasis and bronchoscopic examination urged. The patient was referred to Dr. Chevalier Jackson of Philadelphia who reported as follows:

"Mr. C. was referred to us with the opinion that he did not have pulmonary tuberculosis and also that the right lung was collapsed. Our bronchoscopic examination confirmed both of these opinions. As you will see by the enclosed report of bronchoscopy the collapse was due to the right bronchus being plugged with a white pedunculated tumor which had the appearance of being malignant. A specimen taken bronchoscopically confirmed this tentative bronchoscopic diagnosis. Dr. B. L. Crawford who examined the specimen histologically reported the growth to be carcinoma. As the growth is at the root of the lung in the main bronchus external surgery is not indicated. I could easily remove the mass of the growth with biting forceps, but mutilation of a cancer does not seem to be good surgery, and especially so in this case, because carcinoma of the lung is particularly susceptible to arrest by deep roentgen therapy."

PATHOLOGICAL REPORT

"Specimen consists of several small shreds of grayish tissue, the largest measuring 7 mm. in its greatest dimension.

Histology: Sections show the fragments of tissue to be largely composed of masses of large cells with hyperchromatic nuclei. The cells vary greatly in size and shape and are undifferentiated and many show mitosis. There are small fragments of necrotic tissue present and only a very small piece of stratified squamous epithelial mucosa is attached to one of the fragments of tissue.

There is no question that the lesion is a malignant neoplasm and the cells are considered to be epithelial, but they are not sufficiently differentiated to indicate the type of epithelium from which they are arising.

Diagnosis—Carcinoma."

Patient returned to his home in Birmingham for deep X-ray therapy. This failed to accomplish anything save the alleviation to some extent of the dyspnea which undoubtedly must have become progressive. He died April 14, 1928. A summary of the pathologist's report by Dr. George S. Graham is as follows:

PATHOLOGICAL REPORT

"*Anatomical Diagnosis*:

1—Carcinoma of right bronchus. Metastases in liver, spleen, pancreas, cecum and in bronchial, mesenteric, para-aortic lymph nodes.

2—Atelectasis of right lung.
3—Chronic adhesive pericarditis.
4—Chronic adhesive pleuritis.
5—Pleural effusion (right).
6—Postmortem or agonal intussusception of the ileum."

"*Lungs*—Sections through the tumor show a type cell of epithelial characters. It is rounded or polygonal in shape varying at times to columnar. It is arranged in columns which sometimes show irregular central spaces suggesting lumina. Mitosis occurs but is only occasional. In large areas it has undergone complete necrosis. In one block a thick tumor mass is shown beneath a bronchial lumen. It has replaced all the normal structures. Masses of tumor cells occur within the lumen of a large vessel in one block. The blocks show little of the alveolar structures of the lung. When such are present they are being invaded by masses of tumor cells."

Comment—The histology of this tumor indicates origin from the bronchial mucous membrane."

The mechanism of the atelectasis in this case is quite apparent. A malignant growth starting in the main right bronchus produces an harrassing cough, followed by hemoptysis as the tumor ulcerates. The hemoptysis is repeated and finally either a clot organizes or the tumor growth reaches sufficient size to block the main stem bronchus. Coincident with the general symptomatology of progressing malignancy, the air in the right lung is fairly rapidly absorbed because of the blocked bronchus and the picture of massive atelectasis is found. Bronchoscopic, pathologic, and autopsy examinations confirm the diagnosis.

Thymoma of Lymphosarcoma Type*

By Eben E. Smith, M.D., F.A.C.P., *Lieutenant Commander, Medical Corps, U. S. Navy*

THYMIC tumors are of considerable interest pathologically because of their comparative rarity and difficulty in classification and clinically they are of equal interest because of the diagnostic and therapeutic problems involved. A review of the literature suggests that these tumors are probably more common than recorded case reports would indicate. The following case is therefore reported because it presents several features of unusual interest:

A. M. H. was admitted to the U. S. Naval Hospital, Norfolk, Virginia, February 1st, 1928. Diagnosis: Hyperthyroidism. Chief complaint: Difficulty in breathing, choking sensation in throat, cough, pain on swallowing and swelling in neck. Family history: Negative. Past history: Patient has lived all his life in and about Arkansas and his parents before him have also. He has never seen any goiter in that vicinity. He has always been well at home. Since joining the Marine Corps, the only sick days have been for tonsillectomy, September 8th, 1927, with seven days on the sick list. Had broken arm 1919. No other injuries or operations. Nothing has occurred to him or to his family recently which could be considered a precipitating event. Has lost some weight. Best weight 132 pounds, average 125 pounds. Present weight 122 pounds. Head—Negative. Ears—Negative. Eyes—Negative; states that they feel a little sluggish since onset of present illness. Nose—Negative. Cardio-respiratory—No symptoms referable to this tract prior to present illness. Gastro-intestinal—Negative. Genito-urinary—Had gonorrhea in 1923. Neuro-muscular—States that he has been nervous for a year or more. He is an electrician and sometimes has had to hold a screwdriver in both hands to make it steady enough to use. This has passed off since present illness. Marital—Single. November 7th, 1927 patient was examined prior to transfer and found to be physically fit. On November 12th, 1927 record shows an attack of epistaxis that was controlled by adrenalin. Patient was again examined for transfer, prior to November 23rd, 1927 and was found to be physically fit.

Present illness: Began gradually about two months ago, about December 7, 1927, while on transport en route to Nicaragua. The first thing patient noticed was a choking sensation and difficulty in breathing while lying down. This was not severe and was relieved by lying on his side or sitting up. On December 17th, ten days after onset, patient, while asleep, was awakened suddenly by an acute attack in which he had a sensation of having his breath completely shut off and finally got his breath by sitting up and leaning forward. From this time until about the middle of January, patient was relatively free from respiratory symptoms. He noticed some difficulty following exertion and was a little weak but had very little trouble sleeping. He started to cough during this period but paid no attention to it at first. It was a dry, nonproductive cough and was not relieved by sitting up. The cough became worse, however, and about the middle of January pa-

*From the Department of Pathology, U. S. Naval Medical School, Washington, D. C.

tient noticed that his neck was swelling. After this, the cough did not progress but stayed about the same and the attacks of difficult breathing started again, mostly at night. Sometimes, coughing relieved the respiratory embarrassment. After the middle of January, the cough improved but the neck enlarged gradually, "seemed to grow larger daily" and the respiratory embarrassment became more pronounced. At present he cannot get his breath while lying down and is comfortable only while sitting up with his head forward and he can sleep only in this position. Patient has had no pain except in the muscles of his neck. He states he was nervous before present illness started but has been relieved since. He has noted no changes in his eyes. He thinks that his heart beats faster than it used to, especially when he coughs, and he has a tendency to become a little nervous.

Physical examination: General:—Patient sitting up in bed and leaning forward. He is well developed and nourished, has a worried expression and respiration is labored. There is a swelling at the base of his neck anteriorly. No other gross abnormalities noted. T—99.6. P—124. R—24. Head—Negative. Eyes—Pupils are regular and react normally to light and accommodation. Sclerae are normal. No exophthalmos or lid lag. No failure of convergence. Slight lateral nystagmus. Consensual reflex normal. Ears — Negative. Nose — Negative. Mouth—Mucous membranes negative. Teeth in good condition. Tonsils absent. Throat slightly congested. Neck—Anterior and posterior cervical glands are not felt. Anteriorly above the sternum, the neck is enlarged as by the thyroid. Palpation shows this to be a soft but definite enlargement caused by a mass apparently arising from behind the sternum. It extends upward on both sides of the neck and across the trachea in front of the neck and does not move when patient swallows. The isthmus of the thyroid can be palpated behind the mass when patient swallows. The mass does not appear to be attached to the trachea, larynx, or thyroid gland. Over the trachea, it is about three-quarters of an inch in thickness. Thorax—Normal in size, shape and contour. Normal movement and expansion upon respiration. Tactile fremitus is decreased anteriorly over an area extending about four inches on either side of the sternum and including the heart area. Over the back there is a slight increase in fremitus throughout. To percussion, there is an area of dullness and flatness over the entire presternal area. The breath and voice sounds are somewhat decreased over an area corresponding to the dull area. Expiration is increased, there are many squeaking and sonorous râles all over the chest heard best in the resonant area. Posteriorly, there is a dull note completely across the back above the seventh ribs. Below this line, the note is hyperresonant. There seems to be no corresponding demarcation in the fremitus or breath and voice sounds, all of which are increased. Râles corresponding to those heard anteriorly are heard throughout. The area of dullness shifts with a change in the patient's position. Heart—Apex impulse not seen or felt. Heart cannot be outlined by percussion. Heart sounds are distant, rapid and weak. No arrhythmia. Blood-pressure—Left arm 112/80; right arm 114/82. Abdomen—There is a marked dilatation of the superficial veins. In standing position the superficial veins of the abdomen and thorax become engorged and stand out like cords. No tenderness or spasm. No masses. Liver, spleen and kidneys not felt. Genitalia—Normal adult male. Extremities—Evidence of old fracture of arm. There is no clubbing of the fingers, no ataxia, and no tremor. The veins of the extremities are not dilated. Reflexes—Present and active. There are no hemorrhoids.

February 2, 1928: Basal Metabolic Rate: Plus 95%. Note: "The above findings cannot be considered accurate, but only as an approximation. A technically perfect test is impossible in that the patient cannot lie down, has a persistent cough and the respiration is of the extreme asthmatic type." Complete blood count: Red blood count: 3,850,000. White blood count: 5,700. Haemoglobin 75%. Color index: 0.9. Polys—66; Lymphs—32; Monos—2. Ophthalmoscopic examination negative. X-ray exam-

ination of the chest: "There is a massive enlargement of the mediastinal shadow, so that the shadow extends to the level of the middle third of the clavicle; on the right side the border of the shadow is convex. There are no irregular glandular enlargements outside of the shadow. The lung detail is poor, but there appears to be considerable peribronchial thickening in both lungs. The upper border of the shadow below the 1st rib narrows down and then there is an expansion of the shadow extending upwards into the neck on each side, in the lateral view there appears to be a small space between the anterior limits of the mass and the sternum (Fig. 1). Fluoroscopic examination: "There is a large shadow occupying the mediastinal space. In the posteroanterior view the heart cannot be seen; rotating the patient the heart is located, pushed to the left and backwards. The tumor mass does not pulsate. Impression:—Tumor of mediastinum, probably sarcoma."

February 3, 1928: Diagnosis changed this date to sarcoma mediastinum, and patient was transferred to the U. S. Naval Hospital, Washington, D. C., as an emergency

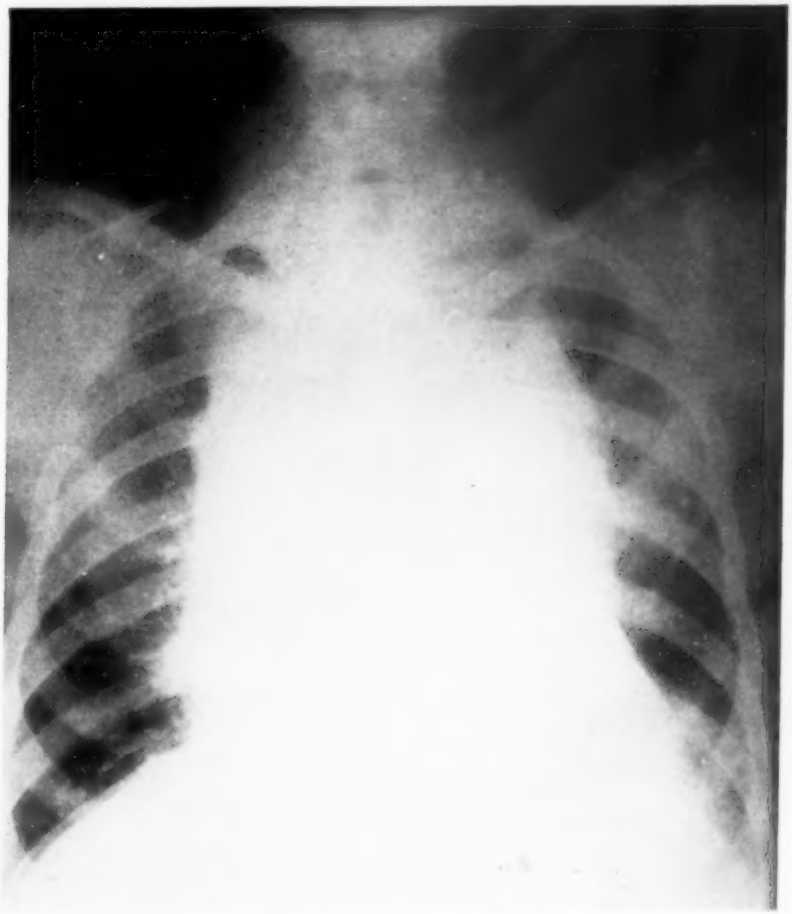

FIG. 1. Taken three days before death; obtained with difficulty due to respiratory embarrassment and lateral view is unsatisfactory for reproduction.

case, for deep X-ray therapy. Patient was admitted to the U. S. Naval Hospital, Washington, D. C., Sunday morning, February 5th and died within 24 hours from cardio-respiratory embarrassment.

Autopsy Report

Autopsy two hours after death. General: Body is that of a white male. Height, 67 inches. Weight approximately 125 pounds. Age, 25 years. Body still retains some warmth. Rigor mortis is absent. There is some lividity, particularly over the sides, chest, around the shoulder girdle, neck and face. Ears are cyanotic. Finger nails cyanotic. No edema. No abdominal distention. Neck is swollen, somewhat boggy, more so on the right. In the mid-line, just above the sterno-clavicular articulation, a tumor can be felt. Superficial veins of chest are enlarged. No definite caput medusae. Axiliary glands just barely palpable. No glands or swelling noted in the supra-clavicular fossae. No cervical glandular enlargement. Eyes—Pupils moderately dilated. Fresh blood draining from nose and mouth. Teeth in excellent condition; all present. One vaccination scar insertion left deltoid.

Chest: On opening chest a large tumor mass is exposed, situated almost symmetrically in the mid-line. Entire mass, including heart, lungs and neck structures, removed intact. See Figs. (2) and (3). Weight of entire mass, Gms. 4400. Net weight of tumor mass, Gms. 3110. Dimensions: Length, 30.0 cm.; Breadth, upper chest, 14.0 cm.; Breadth at base of diaphragm, not including extensions along diaphragm, 22.0 cm.; Depth, at bifurcation trachea, 12.0. Origin not definitely determined, but apparently originating in anterior mediastinum. Mass extends into neck to isthmus of thyroid, infiltrates adjacent visceral and parietal pleura, especially of the diaphragm, and surrounds the heart to the extent that it cannot be identified on anterior exposure. In the upper half of the mediastinum it is intimately adherent to the sternum, but there is no erosion. Posterior wall in mediastinum uninvolved. Tumor is firm, gray and has a lobulated smooth surface. Section shows a homogeneous tissue almost white and friable with no gross evidence of reticulum or fibrous septa and with occasional small hemorrhagic areas of discoloration (See Fig. 4). Considerable clear fluid is present in both pleural cavities. Lungs uninvolved except that tumor has infiltrated the pleura adjacent to tumor, are crepitant, and appear normal except for some congestion. Pericardial cavity distended with increase of clear fluid, and parietal pericardium is granular from tumor invasion. A small area of the posterior wall is uninvolved. Heart distinctly smaller than normal and is flabby. Left ventricle contracted, right dilated. Valves, coronaries and aorta normal. The ascending aorta, the aortic arch, the superior vena cava and the pulmonary vessels are incorporated in the tumor mass. The intra-thoracic portion of the branches of the arch and the branches of the vena cava are also surrounded and apparently compressed and the venous system is engorged. The tumor infiltration appears to invade the adventitia of the vessels but not the muscularis. The esophagus is uninvolved and the trachea and its main branches occupy a depression on the posterior surface of the tumor. They are not infiltrated. At the root of the lungs, however, both bronchi and vessels are surrounded by tumor.

Abdomen: Peritoneum smooth and glistening. Slight, if any, increase in fluid. Omentum normal. All vessels congested. Gastro-intestinal Tract—Nothing unusual noted in stomach and intestines, other than congested vessels. There are many palpable nodes in the mesentery which are small, discrete and relatively soft. A few in the region of the celiac axis are enlarged. The appendix is retrocecal and quite long, measuring 15.0 cm. Liver—Weight, Gms. 1530. Chocolate in color. Liver border reaches costal margin. Surface smooth and glistening. On section, congested. Lobulation is not accentuated. Ducts are patent. Gall bladder contains a few cc. of mucoid bile. Pancreas—Weight, Gms. 90. Appears normal. Spleen—Weight, Gms. 270. Slate blue in color. Rather soft. Capsule is smooth. On section follicles are accentuated. Kidneys—Readily mobilized. Capsules strip

FIG. 2. Anterior aspect of entire mass including lungs, tongue, trachea, thyroid gland and esophagus. Mass was adherent to sternum at point where grey area shows on mass.

FIG. 3. Posterior view of entire mass, includes lungs, trachea, upper section of esophagus, descending aorta and reflection of tumor growth along diaphragm. The discoloration is due to improper fixation.

FIG. 4. To show character of cut section of tumor.

readily, leaving a mahogany-red smooth surface. On section cut surface is distinctly congested, and dark red in color. Pyramids are accenuated. Cortex measures 7.0 mm. Left kidney, weight, Gms. 170. Right kidney, weight, Gms. 150. Ureters are not dilated. Bladder contains a small amount of clear urine. Adrenals appear normal.

MICROSCOPICAL REPORT

Heart: Free from infiltration. There is some atrophy of the muscle cells with deposition of brown granular pigment.

Lungs: Negative except for a fairly marked degree of passive congestion. One section includes area of pleura that is infiltrated with tumor cells. The infiltration is limited to the pleura and adjacent lung field and resembles in type that found in the tumor. There is a mass of the tumor on the pleura the cells of which invade and practically replace the pleura, a few fibers of which are contained in the tumor. The cells infiltrate entirely through the pleural layer and into the alveolar walls and the strands of connective tissue entering the lung from the pleura. (See Fig. 5.)

Liver: Shows congestion, granular degeneration and pigmentation, more marked in the central zone.

Pancreas: Shows vascular engorgement.

Spleen: Structure is relatively normal. Germ centres are large, not very compact, and not particularly active. Centrally some have some nuclear debris. Stroma not increased in amount. Vessels and sinuses

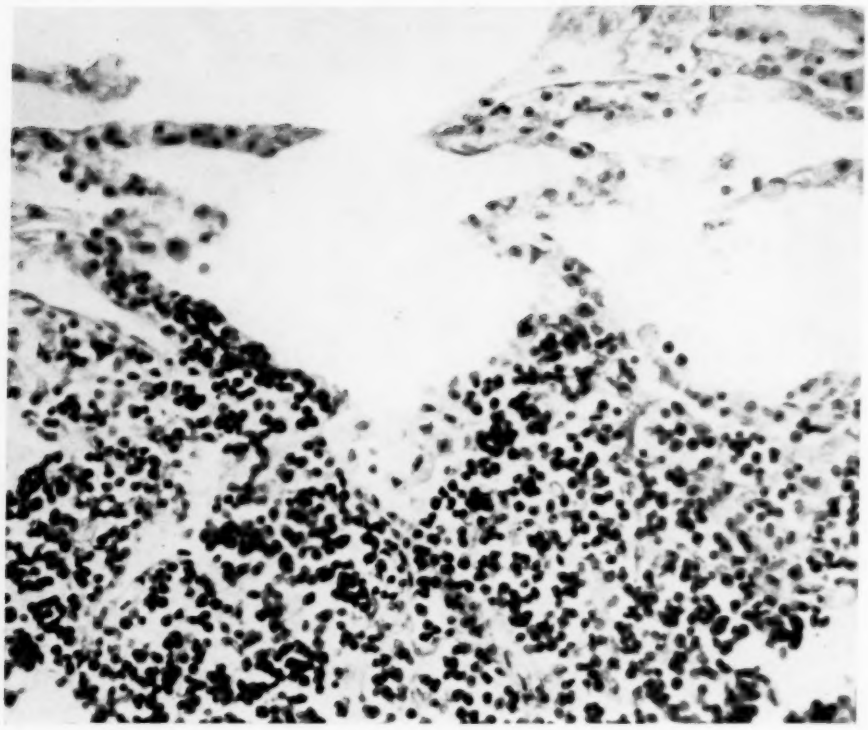

FIG. 5. Photomicrograph of pleural involvement showing character of tumor cells and invasion of pleura and adjacent area. x405.

congested. There is an increase of reticulo-endothelial cells.

Kidney: Shows marked vascular congestion.

Adrenal: Capsule is thickened and has a considerable degree of infiltration with lymphocytes.

Thyroid: Some epithelial hypertrophy is present, but there is no hyperplasia. Some undifferentiated epithelium occurs. There is some lymphocytic infiltration in the stroma.

Tumor: Tumor consists predominately of cells of lymphocyte type with no structural differentiation and relatively little stroma. It is well vascularized and no necrosis is evident. Section through a lobulated mass at the border of the tumor shows tumor growth arranged in a whorl like structure, but here as elsewhere there is no attempt at germ centre formation. The cells consist predominantly of lymphocyte type but there are many immature cells of lymphoblast type diffusely distributed (See Fig. 6). Reticulum is comparatively fine in structure and varies somewhat in arrangement. In some areas it presents an irregular meshwork (See Fig. 7), while in other areas there is tendency for the strands to take a parallel course and for the tumor cells to be arranged along the strands as if attached to a basement membrane. No epithelium or hyaline structure suggestive of Hassall corpuscle formation and no polyhedral reticulum cells or myeloid giant cells, such as have frequently been reported in this class of tumor, are found in any of the sections.

Lymph nodes: Mediastinal node shows a central caseation and peripheral zone cannot be differentiated from tumor tissue. Mesenteric node shows a marked reticulo-endothelial hyperplasia and lymphoid tissue

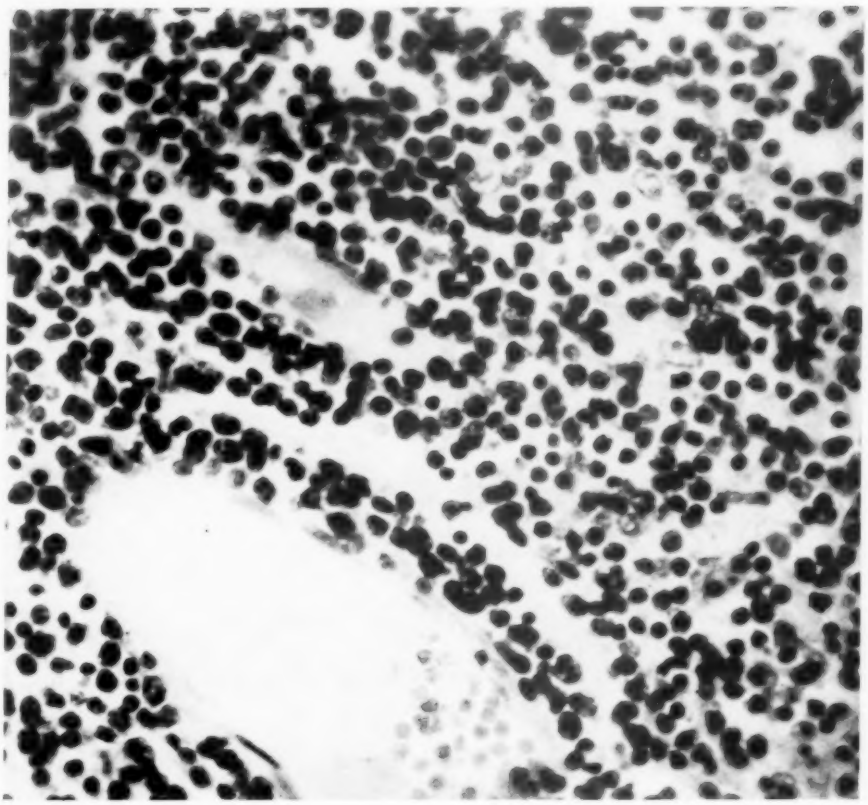

FIG. 6. Photomicrograph of section of tumor showing characteristic structure. x860.

cannot be distinguished from tumor growth except that it is differentiated into germ centres and the immature type of cells is less common.

Pathological Diagnosis: Lymphosarcoma, mediastinal, massive, thymus. Vascular obstruction, great vessels of chest, pressure. Myocardial insufficiency.

Classification of tumors of thymic origin awaits final decision as to the histogenesis of the various types of cells found in the thymus and acceptance of a uniform nomenclature. There is essential agreement that the thymus originates as paired invaginations of entodermal epithelium from the third and fourth branchial clefts. As the gland evolves, embryologically, lymphoid tissue becomes dominant in the picture. According to histologists the original entodermal epithelium persists in the adolescent thymus as (a) reticulum forming a fine meshwork syncytium in which the lymphoid cells are incorporated and as (b) clumps of concentrically arranged flat cells known as Hassall corpuscles. Opinion is still divided as to the origin of this lymphoid tissue. Pappenheimer (1) and others maintain that it originates from the original ento-

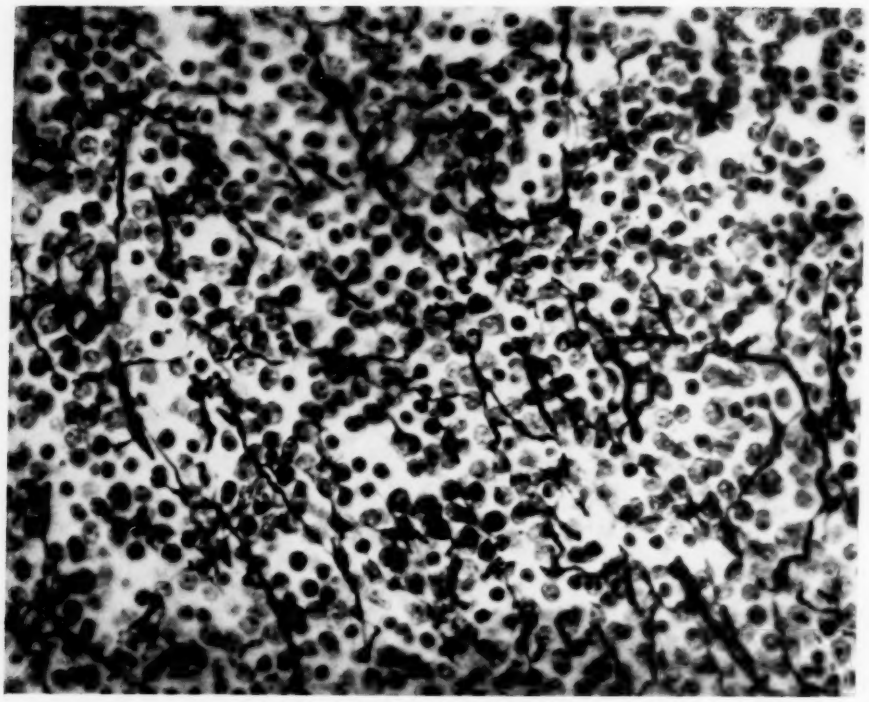

FIG. 7. Photomicrograph, reticulum stain, showing irregular distribution of reticulum and cells. x860.

dermal epithelium. This author suggests the probability that these lymphoid cells may originate from the reticular endothelium even in adult life. Danchakoff (2) Maximow (3) and Hammar (4), among others, maintain that these lymphoid elements are true lymphocytes of mesodermal origin that infiltrate the thymus in early embryonic life.

Whichever opinion is correct, a review of the literature indicates that thymic tumors manifest marked diversity of type. On one extreme is the rare pure epithelioma and on the other extreme is the more common lymphosarcoma with no characteristic epithelial structure in evidence. More commonly, both elements are represented with the lymphoid type of cell usually predominating and the histopathological picture is correspondingly confused. Ewing (5) proposes the following practical classification of these tumors: "(1) Lymphosarcoma or thymoma, composed of a diffuse growth of round, polyhedral, and giant cells. The source of this tumor is probably the reticulum cell, but lymphocytes are present in abundance. (2) Carcinoma arising from the reticulum cells. To these may be added very rare and somewhat questionable cases of tumors attributed to the stroma and called (3) spindle-cell or myxosarcoma."

The interesting features of this case are: (a) The unusual size of the tumor, exceeding that of any found on record. Sternberg (6) reports an unusually large lymphoid tumor of the thymus that in several respects resembles this case. It measured 22 x 13 x 6 cm. and is the largest tumor found on record. His patient was 16 yrs. old. Duration of symptoms prior to death was three weeks which is the most rapidly fatal case found recorded. The pericardium was found infiltrated, firmly encasing the heart. There was a slight but not marked general enlargement of the lymph nodes. His case differs in that postmortem examination of the blood revealed a leukemic blood picture. (b) The type of cell. There is no suggestion of epithelial or recticulum cells, no giant cells are found and lesion does not resemble Hodgkin's granuloma. Cytologically this tumor corresponds most nearly to a pure lymphosarcoma. (c) Tumor was limited to growth by direct extension and invasion of neighboring tissue while present was not marked. The lymph nodes found in the mesentery are not suggestive of metastases. These nodes were definitely hyperplastic but not more so than is observed commonly in conditions where the question of malignancy is not considered. The tonsillectomy performed one month before onset of symptoms may be of significance. The tonsils were not examined histologically. The surgeon noted nothing unusual except that they were hypertrophied and cryptic and patient had a normal convalescence.

The tumor reported conforms with the type usually found originating from the thymus and falls in the lymphoid group which is more common than the epithelial type, differing however, in that the cells are uniform rather than diversified in type and no epithelial structures are included. It extended from the thyroid to the diaphragm and originated in the anterior mediastinum. It was roughly pyramidal in shape, with the base down, had a firm consistency, surface was somewhat lobular but cut surface was relatively homogeneous, surrounding and compressing rather than invading the great vessels, trachea, bronchi and heart and was limited to the thoracic cavity. The clinical manifestations also conform with the usual findings, namely, a relatively rapidly growing tumor causing respiratory and circulatory embarrassment resulting in death approximately eight weeks after onset of symptoms. Two months duration is not rare with this class of tumor but the average is longer. Death within three weeks (6) and five weeks (7) has been reported. A dry nonproductive cough is commonly the first symptom manifested by these tumors and with its onset or later dyspneâ develops. In this case dyspnea antedated the cough but both were present and caused the chief embarrassment the patient suffered.

Development of collateral circulation due to pressure upon or thrombosis of the vena cava has been reported (8), (9), (10), (11), (12), (13) and (14), among others. No thrombosis was present in this case but the pressure was sufficient, on the superior vena cava, to cause a developing collateral circulation. This was quite evident during life but was in-

conspicuous at autopsy suggesting recent onset and only partial obstruction. Cardiac function was further embarrassed by the tumor infiltration into the parietal pericardium which was so extensive as to practically destroy the elasticity of this membrane.

Erosion of the tumor through the anterior chest wall (7), (15) and (16), or of the thoracic vertebrae (17), (18), (19) may occur. It was not present in this case. However, the tumor had to be cut from the sternum and bony erosion would seem a reasonable probability had the patient survived for a longer period. The blood picture was normal. Cases are on record manifesting a lymphatic leukemia, (12), (16), (20) and probably (6) in which the type of leukemia is not specified. Tumors or other abnormalities of the thymus occur in association with myasthenia gravis in sufficient frequency, approximately 50 per cent (21) to suggest that it is not a mere coincidence. A typical case is on record in which a carcinoma of the thymus was found at autopsy (22) and another case responded favorably to X-ray treatment to a thymus tumor (22). These tumors may be confused with Hodgkin's clinically and doubtless have been erroneously diagnosed such. Limitation to the mediastinum should suggest the possibility of a thymic origin. Even histopathologically the tumor may resemble the granuloma of Hodgkin's (5).

There are clinical features that suggest a differential diagnosis between tumors that are carcinomatous and those that are sarcomatous in type. Biopsy is the only positive means of differentiation. Carcinoma usually occurs in later life, after 40, while sarcoma occurs predominantly before 25, suggesting a possible relationship with a disturbance with the normal involution of the thymus which occurs with adolescence. Carcinoma does not tend to reach such large size and is not so rapid in growth. However, epithelial tumors of considerable size may occur (17), (20) and (23). Sarcoma may erode the sternum while involvement of the thoracic vertebrae has been by carcinoma. No definite exception to this localization has been found in any of the case reports. Carcinoma has a greater tendency to metastasis. Sarcoma with an associated lymphatic leukemia is an exception to the tendency of sarcoma to remain localized in the mediastinum. Sarcoma compresses rather than invades the great vessels, trachea and esophagus. Carcinoma is more likely to invade these structures. Consequently thrombosis and terminal hemorrhages are more frequent with carcinoma.

Diagnosis is greatly facilitated by X-ray. Exposure in the lateral position is particularly useful. These tumors, if not large, do not cast a dense shadow and a negative finding may confuse rather than assist in the diagnosis. The prognosis, as with malignancy in general, is unfavorable. Early intensive treatment with X-ray and radium has given some hopeful results and the lymphoid type may be especially responsive (11), (15), (16), (22) and (24). Surgical removal has been suggested but there is no record of this having been done. Palliative relief of pressure upon the

trachea has been obtained by dividing the manubrium and separating it from the sternum (9).

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A Further Experience in the Etiology and Treatment of Encephalitis

By S. R. Salzman, M.D., *Toledo, Ohio*

At this time, when another epidemic of influenza is spreading throughout the country, we may expect again the recurrence of some cases of encephalitis of the so-called epidemic type.

Because of the high mortality and still higher morbidity and the marked economic loss following this disease, everything which may hold out any hope in the treatment of this disease deserves consideration by the medical profession.

This is my excuse for again calling attention to the probable association between gall-bladder disease, or more particularly, gallbladder toxemia, and this type of encephalitis.

Previously I reported (Archives of Neurology and Psychiatry, Nov., 1925, Vol. 14, pp. 638-648) ten cases of encephalitis treated according to the theory that the gallbladder is responsible for this disease, and the results were recorded. Only a few of the outstanding cases were reported in this article. In all, I have had a total of about forty cases, most of them of the endemic or sporadic type.

However, because of an experience during the recent epidemic, I am reporting one outstanding case in order to again call attention to the remarkably clear-cut results obtained by this method of therapy.

The question naturally raised following the report of the first series of cases was: "Are these cases of encephalitis the true epidemic, lethargic encephalitis, associated with epidemics of influenza?"

My comment at that time was that since the text books on the subject scarcely mention encephalitis as a clinical entity, that only two cases had been reported in a search of a number of text-books on neurology, it is probable that most of the cases, if not all seen at this time, are really cases of encephalitis of one type, despite the variations in the severity of the cases.

However, the case presented here occurred during an epidemic and following a rather severe case of influenza, and leaves little room for doubt that it is the well recognized type of epidemic encephalitis.

It may be well to again call attention to certain facts which speak against encephalitis being an infectious, epidemic disease.

These points were noted in the original article:

1st. The evident involvement of the brain by way of the blood stream. Most, if not all, infections into the cerebral spinal axis are by way of the

lymphatics, through the nasal mucous membrane, or nasopharynx.

2nd. The rather prolonged prodromal period present in the majority of the cases. As noted, this prodromal stage cannot always be determined until the patient recovers and is mentally clear enough to give a more accurate history than can be obtained during the acute illness.

3rd. The marked variability in the duration of the illness. Practically all contagious diseases are self-limited. Their duration is fairly uniform in all cases. Encephalitis has no uniformity, no two cases are alike, and the duration may be from a week to months or even years, suggesting rather strongly the continuation of a toxic factor rather than a bacterial disease.

4th. The complete absence of suppuration in any of the reported cases.

5th. The variation in the pathology, the finding of the earliest acute congestive stage along side of the last stages of destruction of nerve tissue and scar formation, indicating again a continuation of the virus and not the uniformity in pathology which is found in such disease as scarlet fever, typhoid, pneumonia, chicken-pox, small-pox, etc.

Case History: Patient, Mrs. M. B. Age 30 years. Seen on November 29, 1928.

History given is that three weeks before she had had a bad head cold. This lasted a week or so, and she recovered without any treatment, and felt fairly well. On November 25, she again developed a cold beginning with a rhinitis, headache, ringing in the ears; felt chilly, and ached all over. By the following day she had some fever and kept in bed, but did not improve.

She was seen by me on Thursday, November 29.

At this time the patient had a temperature of 104; there was definitely present some congestion of the eyes and nose; throat was dry and glazed; and there was evidence of slight pulmonary infection, a few moist râles around the base of the lungs, posteriorly.

The following day, she felt considerably better.

On Saturday, also, she felt much better; but on Sunday she was seized with a very severe headache which became progressively more intense. Her temperature went to 101, and there was apparent some rigidity of the neck.

She was taken to the St. Vincent's Hospital.

The findings at this time, on more complete examination, showed a marked rigidity of the neck, photophobia, slight ptosis of both eye-lids, generalized hyperesthesia particularly marked over the abdomen and legs. The abdominal reflexes were obtained with difficulty and disappeared a day or two later. There was a suspicious Babinski on the right side, and normal reflexes on the left.

The leucocyte count varied between 8,000 and 11,200.

On December 3 she was examined by Dr. Chas. R. King for evidences of involvement of the sinuses, and the following report was made by Dr. King:

"In mydriasis: No blood vessel changes were noted. The media clear; no edema of the retina or disk; fundi

normal. Ears, all landmarks present; no perforations; light reflexes of good quality. Nose, frontal and maxillary sinuses clear to illumination. Mucosa of the nose acutely inflamed; some mucoid material in the left middle meatus, origin undetermined No pus was seen in either nares."

An ex-ray of the head and sinuses was reported normal by Dr. Murphy.

On the third of December a spinal puncture was made. Fluid was definitely under increased pressure; the cell count was 15 and globulin, a trace.

The patient's temperature, the day after admission, went up to 102½, and ranged from 99 to 102½ for several days, finally dropped to 99.6, and did not rise above that during the balance of her stay in the hospital.

She continued to be extremely sick. The rigidity of the neck was constantly present, photophobia and ptosis were marked. The hyperesthesia was particularly distressing to the patient, and a mild nausea was complained of. She could take very little nourishment, as the effort of eating seemed to distress her. She was dazed and was drowsy most of the time, but was easily awakened and responded to questions rather intelligently. Some visual disturbances appeared, and she was unable to read the large headlines on the newspaper, and diplopia was complained of on two occasions.

Under the usual treatment, including salicylates and glucose intravenously, the patient did not get any better.

At this time it was impossible to make any definite diagnosis of gall-bladder involvement. She was hyperesthetic and tender everywhere, and the tenderness was just as marked on the left side as under the right costal border, and had it not been for my previous experience with this problem, I should have found no excuse for further investigation of the biliary tract; but because of this experience and because she did not respond to any treatment but was definitely getting more and more drowsy and seemed to be suffering considerably, an attempt was made to pass the duodenal tube on December 10.

This attempted drainage was not successful. The tube was passed again the following day, and a very successful drainage was obtained of dark bile, rich in Gram positive cocci, and showing many epithelial cells both of the columnar and squamous type.

The following day the patient showed very marked improvement. The ptosis had disappeared, the rigidity of the neck was much less marked, and mentally she was perfectly clear, able to sit up in bed and feed herself, and she stated that she felt well enough to want to sit up in a chair.

Drainages were repeated again on the 12th and 13th, with continued improvement, return of the abdominal reflexes, and disappearance of the Babinski on the right side.

The clear-cut result in this case leaves little room for doubt in my mind and in the minds of the medical men around the St. Vincent's Hospital who saw this case, that it can be attributed only to the biliary drainages instituted and the consequent lessening in biliary infection or toxemia.

The microscopic findings in the duodenal contents removed by the tube

from cases of encephalitis have been quite variable. Many have been clear and without any remarkable microscopic picture. Usually the fluid is very dark and sticky. It changes very promptly, within a moment or two after removal from the bile tract, to greenish bile showing very early oxidization process.

Occasionally there are very large numbers of epithelial cells, squamous and columnar in type, with extraordinary numbers of Gram positive organisms, usually a coccus, very small in character, the cultural characteristics of which I have not worked out. Occasionally a very short, thin, Gram positive rod is noted in considerable numbers; and now and then the bile, as it comes out through the tube, is noted to be of an oily consistency. In other words, nothing characteristic is noted in the character of the fluid.

It is not my contention that the gallbladder shows any degree of infection. Rather do I feel that we are dealing with a toxic state; that whether bacterial infection was the primary cause of the condition or not, we are dealing either with the metabolic product of the bacteria in the gallbladder, or, more probably, with chemical changes in the bile itself, so that there is absorption of toxic products of a neurotrophic character into the circulation.

The possibility of an allergy, also, must be borne in mind in this type of condition. This possibility is somewhat heightened, too, by the occasional finding of a very marked improvement in the encephalitic condition, following the appearance of an extensive skin rash.

However, this subject needs considerable study before any definite conclusions can be drawn concerning it. Whether the condition be directly shown to be a toxemia or an allergy, the toxin or the sensitizing substance is probably located in the bile tract.

I do not mean to imply that all cases of encephalitis are associated with or caused by biliary tract pathology. We know that encephalitis occurs in a number of other conditions, such as various septic states.

I have reported, in the past, encephalitis following sinus infection, and dental infection, and have recently seen a very marked and very severe case following pelvic operation in which there was probably thrombosis of the pelvic veins. The fact that a similar type of encephalitis can occur in a number of conditions giving rise to toxemia also heightens the probability that the encephalitis following epidemics of influenza is of the same type, i.e., toxic.

My earlier experiences led me to feel that the majority of cases require surgical treatment. Experience with a larger number of cases requires revision of this statement. The majority respond well to non-surgical biliary drainage.

The indications for surgical treatment are:

1st. Failure to pass the duodenal tube, either because of lack of cooperation or comatose condition of the patient.

2nd. Failure to get bile on two or three successive trials provided the tube is definitely in the duodenum.

3rd. Conclusive evidence of cystic duct obstruction; i.e. failure to secure

"B" bile, or x-ray evidence of dysfunction of the gallbladder.

Of my forty cases, fourteen were treated surgically. A larger series may show a still smaller percentage requiring radical treatment, especially if the condition is recognized early and proper treatment is instituted before the patient becomes too comatose to swallow the duodenal tube.

It is certain that the experience reported here and in previous articles is worthy of the serious attention of the medical profession throughout the world in the hope that the observations reported by me may prove of real value in the treatment of this very serious and apparently hopeless disease.

The Histophathology of Appendiceal Amebiasis With Case Report*

By Winston F. Harrison, M.D., C.M., *Montreal, Canada*

THE case to be reported is of interest first of all because the patient had never been farther south than the State of Michigan. Secondly, the case presents some features rarely found even in districts where the disease is prevalent. Lastly, it is peculiar in the mode of diagnosis, which was made by study of the vermiform appendix in the course of the routine histopathological examination of material from the surgical clinic. Since the first reported case of amebic dysentery in the State of Michigan by Dock[1] in 1902 there have been from time to time reports which indicate that though the disease is not common it cannot be considered a great rarity in northern latitudes. The great majority of cases met with in temperate zones, however, give a history of having been in the tropics at some period of their lives. Symptoms of the disease have then appeared after a variable length of time, often after many years of residence away from tropical countries.

Case Report

N. K., a white male, age 40, a millwright by trade was admitted to the Medical Outpatient Department of the University of Michigan Hospital, on July 31, 1928, complaining of ache and soreness in the abdomen. His present illness he dated to two years before, when he began to tire easily and to lose weight. Later, he had pain in the right lower abdomen coming on shortly after meals. For a time he had a "bloody diarrhea." The blood appeared after the bowel movement. He had suffered with piles for about 4 years. After the first attack the patient improved, but the symptoms recurred at intervals of 4 to 6 weeks. About two months before admission he began to have a constant soreness in the right upper abdomen. Two weeks later his local physician found a lump at the site of the soreness and made a diagnosis of hernia. The patient was advised to get a truss. The truss made the soreness worse. Since then he had had a steady ache in the right side. He began to lose appetite and to lose weight. He was unable to work because he felt weak and tired. The diarrhea had not been constant. At times he was slightly constipated. He had no nausea or vomiting, but had lately been much troubled with gas. He had lost 37 pounds in weight during the 13 months before admission. Three weeks before admission he noticed a swelling of the peri-anal tissue. Since this time he had some difficulty in defecation, and occasionally incontinence of feces followed by the passage of small amounts of fresh blood and yellowish material. He had never noticed tarry or clay colored stools.

Past History. The patient had always lived in Michigan. His general health had been fair. He had measles and mumps in childhood. At the age of 2 years he had

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an umbilical hernia, and at 9 a left inguinal hernia, but was not operated upon. He stated that he had had loose stools nearly all his life. From childhood until he was 18 or 19 years of age he had "ulcers of the bowel." No other relevant history was obtained at this time.

Family history was essentially negative.

Physical examination showed an emaciated male with good muscular and skeletal development lying comfortably in bed apparently without pain. The abdomen was slightly below the level of the thorax. Panniculus was scanty. The abdomen moved freely with respiration. Just below and to the right of the umbilicus a mass about the size of a turkey egg was felt. It had a definite border on the left and was nodular, firm and fixed. It was somewhat tender on palpation. Other parts of the abdomen were soft, and there was no splinting of the recti. The liver and spleen were not palpable. There were several tags of mucous membrane protruding from the anus. The rest of the examination was essentially negative.

While still attending the out-patient department he was referred to the Department of Surgery. The impression at this time was that he had a carcinoma of the cecum. He was admitted to the Medical Ward on August 16 where a more thorough examination was made. The X-ray findings were as follows. The first report stated that the appendix was visualized, movable and not tender at 24 hours and empty at 48; the cecum and colon were poorly filled with the barium meal, and had a smoothed out appearance. Later, a barium enema was given. The findings at this time are well shown in the accompanying plate. (See fig. 1.) There was a definite obstruction to the flow of barium in the mid portion of the transverse colon. The cecum and ascending colon filled very poorly and showed irregularities of outline. A diagnosis was made of carcinoma of the colon in the region of the hepatic flexure. The chest plates showed no evidence of pulmonary disease.

The urine examination was essentially negative. The blood showed 4,440,000 reds, 9,350 whites and 60% hemoglobin. The stools (at this time 4 to 6 a day) were reddish brown, liquid, and showed considerable gross blood. There was a positive guiac test. There were no other positive findings in 11 stool examinations. The patient's temperature ranged from normal to 99°, and the pulse from 80 to 90 during his two days on the Medical Ward.

A consultation was held with members of the Surgical staff. In view of the history and findings, and particularly the X-ray studies showing a very definite obstruction of the large bowel a provisional diagnosis of carcinoma of the colon was made. Operation was postponed a few days because the patient had a slight respiratory infection. On August 22 he was operated upon by Dr. F. A. Coller under nitrous oxide anesthesia. A large mass was found involving the ascending colon, the hepatic flexure and part of the transverse colon. The bowel was injected, and the appearance was that of a massive inflammatory infiltration. The cecum and terminal ileum around the ileocecal valve were injected, thickened and indurated. Retroperitoneal lymphnodes were greatly enlarged. The appendix was remarkably thickened and infiltrated. The mass itself was too extensive to be removed. It was impossible to attempt an anastomosis or short circuit operation because of the condition of the large bowel which showed inflammatory thickening throughout its entire extent. Consequently an ileostomy was done. The appendix and small inflamed lymphnodes were removed.

A pathological examination was made of the appendix by Dr. C. V. Weller in the course of the routine examination of material from the operating rooms. An ulcer in the wall of the appendix contained amebae. (The full report will be given below.) Regional lymphnodes showed a severe lymphadenitis.

In view of finding amebae in the appendix the patient's history was re-taken in part. He stated that he had had bloody diarrhea off and on since childhood. He thought he had piles. Two years before admission he was seized with a cramp in the right side, and had bloody diarrhea

FIGURE 1. X-ray plate showing narrowing and large filling defect in the upper colon. The irregularity of outline in the descending colon is suspicious of the presence of amebic ulceration.

which persisted for 6 months. At that time he was associated with a man from Louisiana who had suffered with dysentery for 6 years. He drank out of the same cup, used the same toilet, but did not live at the same house. His diarrhea improved and he was fairly well until 4 months before admission, when the pain and diarrhea returned. Soon afterward he noticed a lump in the abdomen. After that time he became steadily worse. The patient stated that he had never been out of the State of Michigan except once, and that was to go to South Dakota for a short time.

Repeated examination of the whole stools finally revealed many amebae, actively motile. The type was *Entameba histolytica.*

Two days after operation the patient's condition became worse. The abdomen was distended and there were signs of consolidation at the base of the right lung. The following day, August 25, he died. Unfortunately an autopsy could not be obtained.

Discussion

It is not the intention to give here an account of amebic dysentery, but only to point out briefly some features of the present case and to give some account of the disease in the appendix.

Here then is a case which presented such prominent features of malignant disease that the real etiological factor was unsuspected until the pathological examination of the appendix gave the clew. Had the patient come under observation in a locality where amebic dysentery is prevalent the diagnosis would in all probability have been made earlier. The physician in the tropics comes to suspect amebiasis in cases presenting symptoms of intestinal disease until it can be ruled out The malady is much less common with us. The Michigan Department of Health could give little information as to its incidence because it is such an infrequent cause of death in this State that it is not classified separately. It was stated that cases are rare, and almost invariably imported from the south. Nevertheless within the past few years the number of cases seen in the Temperate Zone is steadily rising. Recently, Williamson, Kaplan and Geiger[15] made a survey of amebic dysentery in Chicago They conclude that the disease is not uncommon in that city, and has frequently been overlooked. So it must be remembered that it is a cosmopolitan disease, and by no means confined to the tropics. At the time Hermann[2] (1919) reported two cases in this hospital there had been a recent collection of 500 cases in this country observed in a latitude corresponding to our own. The dissemination of the disease was much increased during the war by the movement of troops to and from tropical areas.

It is now well recognized that the *Entameba histolytica* is present in the large intestine of many people showing no symptoms of the disease. These amebae were found in the stools of 3% of all American troops, and 10% of those returning from France were similarly infected.[3] Dobell[4] says there is good evidence to show that from 7 to 10% of the population of Britain are infected with *E. histolytica.* Yet the number who show symptoms of the disease is very small. The same authority states that up to 1921 there were scarcely a dozen authentic cases of indigenous amebiasis reported in Great Britain. As far as is known the parasite is identical with that in the tropics. Just why it should

be so much more a tropical disease does not appear to be clearly understood. Not only does the incidence vary but the clinical manifestations are somewhat different. In countries away from the tropics the disease is more chronic. Boyers[5] says that the behavior of cases in the Temperate Zone suggests that the ameba meets with a number of adverse conditions that help to create a subtle picture of chronicity. He adds, "Whether this tendency to chronicity in large numbers of persons in the Temperate Zone means an actual relative immunity is not known." This individual had never been south of the State of Michigan. How did the patient become infected? A point in the history which was finally elicited suggests the answer. For some time previous to his severe attack two years before admission he was associated with a man from Louisiana who was known to have suffered from dysentery for 6 years. If we assume that the man from the south was a carrier it is not unlikely that the patient was infected through him. This would seem to be the most probable explanation in view of his previous good health, unless we are to take into account the somewhat vague history of loose stools and "ulcers of the bowel" in childhood. Man can become infected with *E. histolytica* in only one way, and that is to swallow the cysts of the organism. Authorities believe that the carrier is the biggest factor in the transmission of the disease, and that contaminated water supply plays a small rôle if any. The cysts are ingested by the accidental contamination of food or drink by minute particles of feces containing them.

In the present case it is interesting to note the things which pointed to intestinal malignancy. First, there was the history of the presence of a lump in the abdomen appearing gradually; diarrhea, and at times constipation, with bloody stools; indigestion, loss of appetite, progressive loss of weight and strength. This, together with the finding of a palpable firm mass; the irregular filling defect shown in the x-ray plate; blood and mucus in the stools; the presence of anemia with only slight leucocytosis; and practically no elevation of temperature. At operation the surgeon considered that the tumor mass together with other parts of the large bowel and the retroperitoneal tissues had the characteristics of a massive inflammatory infiltration rather than those of neoplasm. It is important to note that the terminal ileum at the ileocecal valve was also involved.

This type of lesion in intestinal amebiasis is apparently very uncommon. In 1925 Runyan and Herrick,[6] working in the Panama Canal Zone reported 4 cases very similar to this showing "amebic tumors." They state that the condition had not until that time been reported in the literature on amebic dysentry. They describe this type as a massive ulceration due to a chronic amebic infection causing enormous thickening of the wall of the bowel. The condition affects the upper large intestine, principally the cecum, and also, as was shown in all four of their cases, the terminal ileum. The first two were tentatively diagnosed tumor of the kidney and

carcinoma of the cecum respectively, while the last two, "as the result of increasing experience" were correctly diagnosed. In a paper on x-ray diagnosis of intestinal amebiasis Dr. J. J. Vallarino[7] describes the roentgenological findings in one of the four cases. The infiltration had almost entirely closed the lumen of the transverse colon. The patient had every indication of a severe and rapidly growing intestinal malignancy. Whether in the present case the narrowing of the lumen of the bowel was entirely due to infiltration or if there were also strictures from contraction of the gut is not certain in the absence of anatomical evidence. Each condition is reported to occur, though very rarely in the course of chronic intestinal amebiasis.

In the literature on amebic dysentery the vermiform appendix appears to have been singularly neglected by most writers. Councilman and Lafleur[8] in their classical monograph, while giving detailed reports of autopsied cases did not mention the appendix. Most text-books of Pathology either fail to mention it or say that it may be involved, though rarely. Sandwith,[9] in his Lettsonian Lectures (1914) on amebiasis speaks of affection of the appendix only once, stating it to be a very rare complication. Several authorities, however, particularly in recent years, have shown how frequently the organ is involved and its importance especially in surgical amebiasis, and also its relation to the question of carriers and re-infection. Harris[10] in 1898 was the first to report a case of amebic appendicitis although Kartulis, working in Egypt years before, had mentioned finding amebae in smears of contents of the appendix removed post mortem. Schaudinn (1903) who classified the parasites and identified *Entameba histolytica* as pathogenic for man found amebic appendicitis in rabbits in the course of his experiments. Rogers,[11] after much experience with the disease in India concluded that the appendix is often severely affected by the amebae, and instanced cases of fatal peritonitis following perforation. The most extensive work in recent years appears to be that by James[12] and his associates and by Clark,[13] all working in the Panama Canal Zone. They give some prominence to the incidence of appendiceal amebiasis. Clark found that in 186 cases at autopsy 76 had amebic ulcerative appendicitis and 9% had either perforation or abscess formation. He considers that owing to the structure of the organ and that fact that the lumen tends to become more or less obliterated in adult life it may readily become a "carrier pocket" and difficult to treat successfully. These authorities and others now remove the appendix in some instances where medical treatment has been unsatisfactory. Last year Coates and Groves[14] in England, coming to the same conclusions reported 3 such cases very successfully treated by operation.

Cole and Heideman have recently reported a case of amebic ulcer of the abdominal wall following appendectomy with drainage. They found amebae in the ulcer, but although "unusual bodies" were found in the wall of the appendix they were unable to demonstrate the presence of parasites here.

and thus to prove the starting point of the lesion on the skin surface In their conclusions these writers intimate the "possibility that amebas may influence the pathologic process in the appendix in some cases of appendicitis." This is no longer a matter of doubt. There are abundant references in the literature showing that the parasites themselves are sometimes responsible for lesions in this organ, and that amebic appendicitis is a well recognized condition.

We now turn to the pathological findings in the present case.

Pathology of Appendix

Gross pathology:

The appendix is of normal length, but averages about twice the normal thickness. Toward the tip the wall is thinned over a dilated lumen containing a soft material. The surface is roughened by adhesions and by a small amount of fibrinous exudate. On cutting across the appendix the lumen is found to be somewhat dilated throughout the entire length, and markedly dilated in the distal third. Mucopurulent material escapes from the cut lumen. There is no evidence of neoplasm.

Microscopic pathology:

The appendix was examined by microscopical sections cut at 3 levels representing the junction of the quarter segments of the organ. The most distal section shows a very marked dilatation of the lumen with a small amount of mucopurulent exudate still remaining. This has apparently been the site of a fecal concretion since there is a marked pressure atrophy and chronic catarrh of the mucosa. The wall shows fibrosis, with mononuclear and polynuclear infiltration including many eosinophiles. The subserosa shows a marked fibrosis with mononuclear infiltrations and numerous peritoneal adhesions.

Microscopic sections taken from the mid point of the appendix show a moderately dilated lumen. There is a marked chronic catarrh of the mucosa and the same evidences of a chronic appendicitis as found in the section first examined. At this level the serous surface shows a small amount of unorganized fibrinous exudate. Interrupting the mucosa at one point there is an ulcer which extends to the inner circular fibres of the muscularis propria. The greater part of this ulcer lies in the lymphoid tissue of the submucosa. It is bordered in part by a very slight amount of vascular granulation tissue, but for the greater part by the structural elements of the appendix itself. The neighboring tissues are infiltrated to some distance in each direction with polymorphonuclear leucocytes. The ulcer shows an overhanging mucosal edge surrounding its ragged undermined margin. Its base is covered with fresh purulent exudate in which there are numerous amebae to be described later.

The most proximal section from the appendix shows a dilated lumen surrounded by a greatly thickened wall. The general tissue changes are the same as previously described although the purulent infiltration in the wall is somewhat more extensive and there is more fibrin on the peritoneum. About one-fifth of the mucosal surface is the

site of an ulcer. (See fig. 2.) This ulcer extends slightly into the circular portion of the muscularis propria, is slightly concave at its base following the contour of the circular muscle, and at its border undermines the mucosa leaving an overhanging, inwardly rolled shelf of mucous membrane. (See fig. 3.) The ulcer is much larger than the one seen in the preceding section, and at its base there is a leucocytic infiltration extending practically through the appendix wall. The base is covered with fresh and old pus, fragments of necrotic tissue and partially hemolyzed blood clot. Scattered through this material there are numerous amebae, but the parasites are especially numerous in the lower part, the exudate adjacent, or in contact with the living tissue.

The amebae:

In the fixed and stained preparations the amebae average about 60 microns in diameter. In shape they are roughly spherical with a slight blunt lobation. Many show an irregularly crescentic narrow zone or more lightly staining and less granular ectoplasm about the main mass of cytoplasm. The endoplasm stains a medium purplish blue with hemalum and eosin and contains numerous irregular granules, fragments of ingested nuclei, many vacuoles, and in most instances red blood cells in various stages of hemolysis. (See figs. 4 and 5.) The nucleus is in most cases excentric in position and is about 10 to 12 microns in diameter. It varies in staining from red violet to purplish blue, in most instances taking the eosin to a sufficient extent to give it a reddish coloration. Toward the periphery of the nucleus, deep blue to bluish black chromatin material can be seen in small crescent-shaped masses in some cases. From these characteristics, particularly the very general ingestion of red blood cells and the presence of these organisms in an ulcerative lesion, recognition of this ameba as *Entameba histolytica* (*Locschia coli*) seems to be fully justified.

Summary

1. A new case of chronic amebic dysentery is reported in a man who had never been south of the State of Michigan.

2. The case showed such features of malignant disease that a tentative diagnosis of carcinoma of the colon was made. The diagnosis of amebic dysentery was made during the routine examination of the appendix following operation, on the pathology of the ulcers and the finding of the amebae.

3. Appendiceal amebiasis is not an uncommon complication of the disease It is now considered to be important as a "reservoir" in chronic cases and in carriers. Appendectomy has been successful in cases not responding well to medical treatment.

4. A description is given of the histopathology of the appendix in this case.

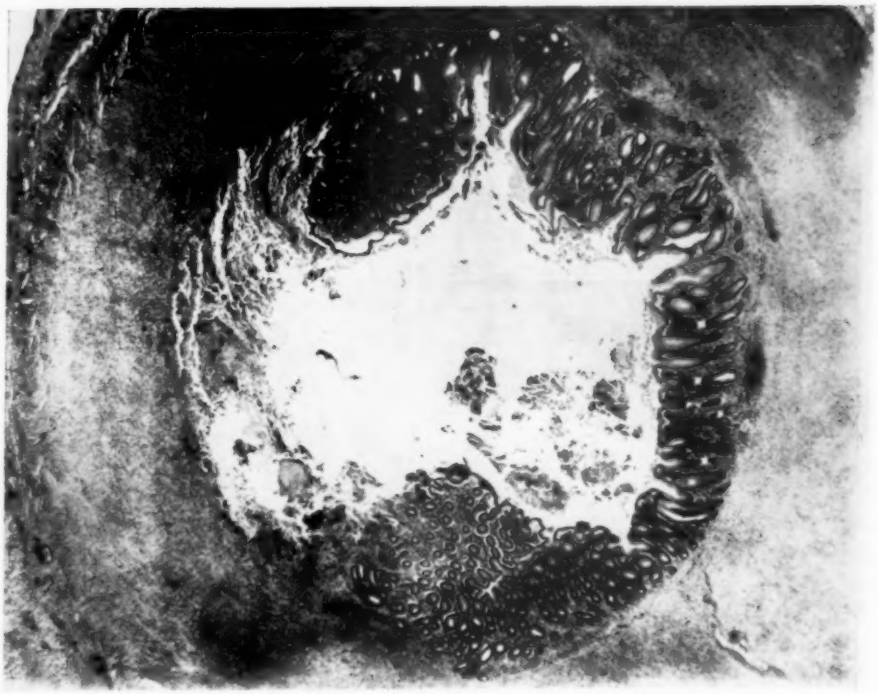

FIGURE 2. Low power photomicrograph of appendix showing large amebic ulcer.

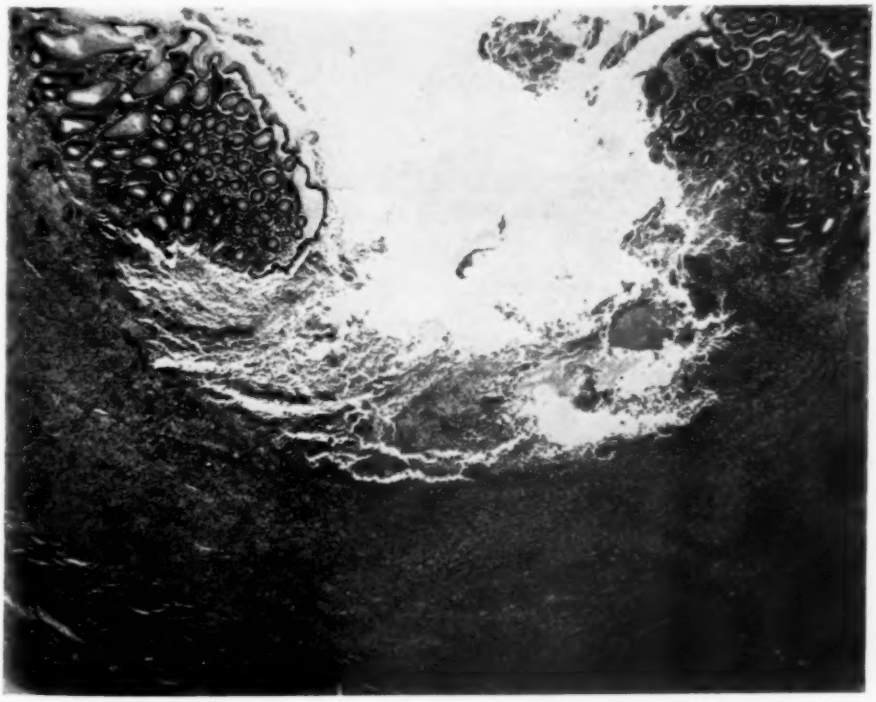

FIGURE 3. Higher power. Amebic ulcer in appendix.

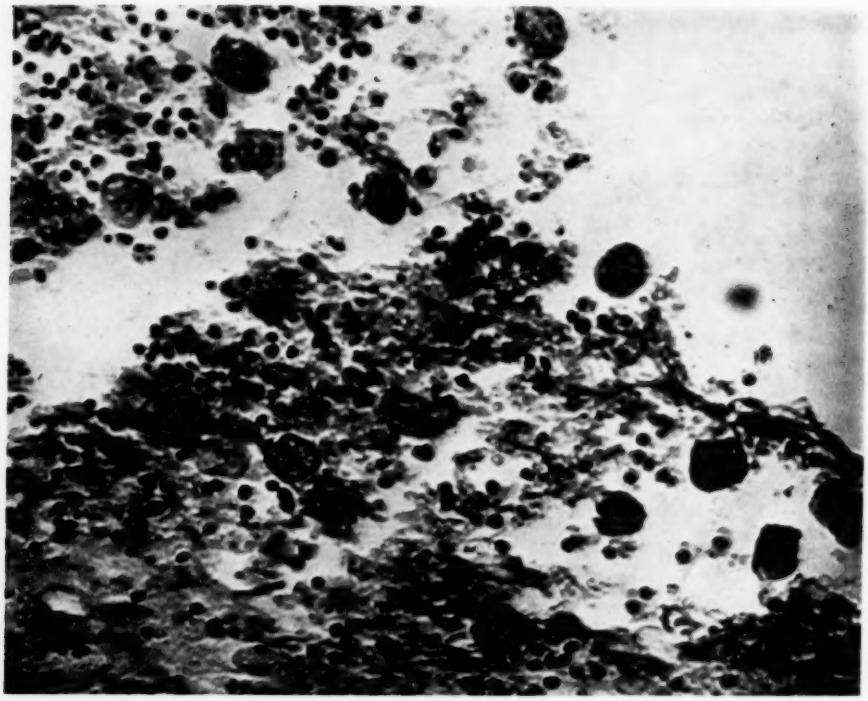

FIGURE 4. High power. Showing amebae at the base of the ulcer.

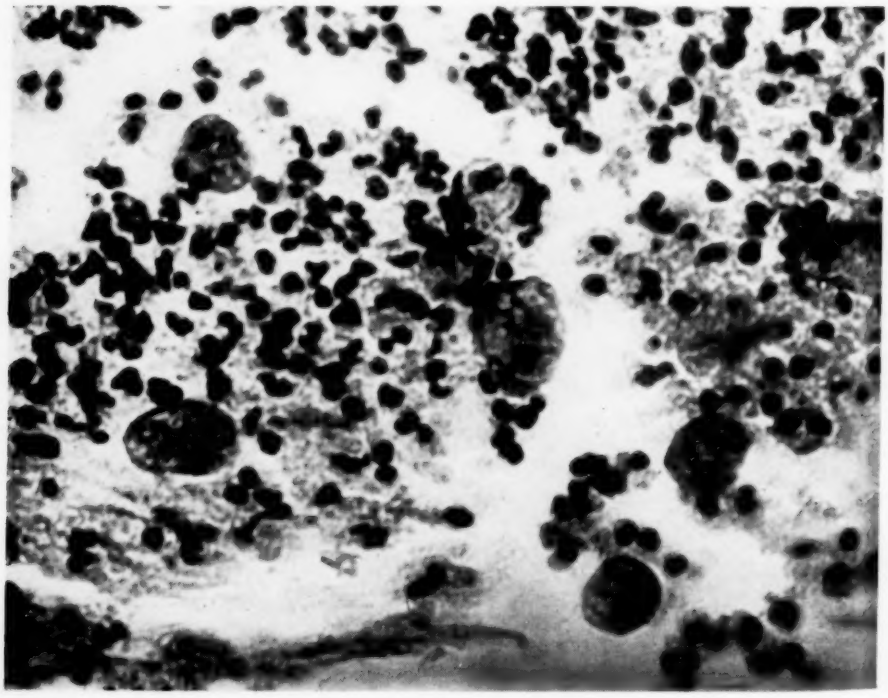

FIGURE 5. High power. Showing detail of amebae.

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Tularemia*

A Consideration Based on a Resume of the Literature and Personally Observed Cases with a Report of an Unusual Complication.

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Introductory

Several interesting features attach to this relatively new disease in man. So far, its incidence has been confined to the United States and Japan, and while it would seem reasonable to expect it to be found wherever certain rodents exist, it has not been recognized elsewhere outside of our country. Second, it was discovered in the course of a general study of diseased rodents. Third, it occurs in nature as a highly fatal disease of certain animals, while recovery is the rule in man. Fourth, within one year after the disease was recognized, the causative organism was identified. Fifth, both were discovered by the same investigators. Sixth, its identification and early study is to be credited to the Public Health Service. Seventh, nothwithstanding its relative infrequency in man it possesses high developmental capacity when accidentally inoculated. Eighth, the evolution of our present knowledge has been wrought by trained workers in special fields—a public health worker discovered the disease in animals, a clinician recognized and described the first human cases, a serologist found the antibodies in his own blood and a bacteriologist recovered the organism from an ocular infection in man. Ninth, the organism was named from the county where the diseased animals were first found. Tenth, despite the short period between its recognition as a separate disease and the discovery of its cause and despite the prompt publication of the results of studies, ten years elapsed before the rodent disease in California and Utah deer fly fever in man were recognized as the same disease, though both were studied in the same year—1911.

It was called Tularemia by Francis and may be described as a specific plague-like infectious disease occurring in nature in certain rodents with occasional transmission to man in a milder form and is due to the Bacterium tularense.

It was originally found in the Western states by McCoy and Chapin in 1911. Since that time it has been found occurring in every section of this country except New England, Wisconsin and Washington and singularly has not, as yet, been found out-

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side of the United States except in Japan.

Mode of Transmission: McCoy, in 1911 published a description of a disease found among ground squirrels in California. The disease was discovered during a systematic examination of rodents carried on at the Federal Laboratory, San Francisco. He demonstrated the transmissibility of the disease to mice, rabbits, gophers, guinea pigs and monkeys, but failed to isolate any organism from the plague—like lesions so produced. The next year he and Chapin isolated the specific germ and were able to grow cultures on egg-yolk medium. By this discovery a new disease was established; the organism was named Bacterium tularense, from the county of Tulare and the disease has come to be recognized in the literature as Tularemia. Cases of infection in man began to be recognized. Chapin became a victim of accidental laboratory infection. The six laboratory workers connected with the public health investigation from 1920 to 1927, likewise contracted it. Similar laboratory infections have been reported notably by Ledingham and Fraser. The Lister Institute of Preventive Medicine, London, exhibiting a high degree of scientific curiosity procured a culture of Bacterium tularense from the Hygienic Laboratory, Washington, D. C., with which they infected guinea pigs. Three of the workers fell victims to the disease, their ardor cooled and the experiments were discontinued. The spontaneous development of the disease in man began to be noted and recorded until now, it has been recognized in every section of the U. S.

Insects play an important rôle in the transmission of the disease. The deer fly, the wood tick, the mouse louse, the stable fly and squirrel flea have been shown capable of such transmission in the laboratory. Game birds, as in the case of the grouse, apparently have been carriers. Handling infected animals has been the usual method of infection, the bacterium finding entrance through an old or fresh wound on the hands. More disturbing than the tick and fly transmission or direct inoculation, is the possibility of transmission by undercooked meat of infected animals as indicated by Freese. Another possibility is by inhalation when laboratory workers handle animals with extensive pulmonary involvement as seems to be established by Parker and Spencer. The same authors have demonstrated that the germ can be transmitted by such intermediate hosts as the wood tick; the eggs, nymphs and larvae may carry the specific bacterium from the parent. If this holds true under conditions outside the laboratory, it becomes at once apparent that when a species of insects become infected, the number of such infected insects may assume tremendously greater proportions than if infection of adult insects occurred only by feeding on diseased animals.

Inoculation from man to man has not been reported and Francis thinks this does not occur. If this method of transmission did obtain, it would be expected that doctors would have been almost certain to have become infected, especially during the earlier period when the disease was not generally recognized, and would now

come to light. Harris has reported a case, however, that indicates the possibility of contact infection of nurse or physician. A mother while dressing the initial ulcer and a wound resulting from incising a necrotic gland on a child, infected her own hand, the skin of which was injured by a safety pin and was followed by a typical glandular type of the disease. While all of the cases seen by me have been traced to infections from dressing rabbits, I can see no reason why injuries to the skin received while dressing human ulcers or wounds following incision of necrotic glands may not become portals of entrance of the specific bacterium.

No instances of infection, by natural means, in domestic rabbits grown in hatches have been reported. Artificial infection can, of course, be established. The wild cotton tail rabbits, snow shoe rabbits and jack rabbits, have all been found infected in nature. The rabbit tick and the rabbit louse are the usual means of transmission in the rabbit family, and, fortunately, neither of these insects bites man. The various blood sucking fly families and the wood tick are the usual carriers from animals to man. Another source of transmission has been added to the list of rodents already known to be carriers of the disease, through Perry's report of the discovery of naturally infected field mice in California. Field or meadow mice are often infected by mites. These mice live in nests and migrate for food and it appears probable that tularemia is transmitted among them by the mites. The bacterium has been demonstrated to be capable of surviving the winter in the tick and may be transmitted in this insect from generation to generation. The bed bug, not to be outdone by the louse, has joined the ranks of possible carriers. Since the squirrel flea carries the disease from ground squirrel to ground squirrel, it is not unreasonable to suppose the pulex irritans may vie with his cousin and increase man's likelihood for infection.

It is one of the most transmissible diseases in laboratory experience. Chapin's accidental inoculation in 1911 might not be considered as unexpected but the number of laboratory workers since 1919 that have been infected can be explained only on the basis of high transmissibility. They were all trained laboratory people and in addition, knew the great risk, hence, took unusual precaution to protect themselves. When these facts are borne in mind, one ceases to wonder why handling or dressing rabbits results in human infection but rather why more are not so infected.

Of forty-nine human cases studied by Francis, fourteen were inoculated by flies, one by ticks, ten had dressed jack rabbits, ten had dressed cotton-tail rabbits and fourteen were laboratory workers who had necropsied infected laboratory animals. That twenty-eight and five-tenths per cent (28.5%) of the total cases Francis tabulated should have been inoculated in scientific investigation constitutes an amazing testimony of the danger of this disease to the laboratory workers.

The Public Health Service has emphasized the tick, fly and dressing wild rabbits as the more frequent sources

of infection. From a seasonal standpoint, the importance of each of these will vary in different localities, hence the disease may be termed endemic, for it may be found somewhere in the United States alone in practically every month during the year.

Pathology: Only four cases of tularemia in man have been studied at necropsy up to November 1927. The first was reported by Verbrycke in 1924, the autopsy being done by Sellinger. This case died eighteen days after the onset of illness and eight days following an exporatory laparotomy. The diagnosis was made after the laporatomy but before death by the agglutination test and cultures from inoculated guinea pigs, rabbits and mice done after the necropsy. Nodules ranging in size from a shot to a walnut were found in the lungs. The spleen was soft, the pulp being almost semifluid in consistency. Circumscribed yellowish white nodules were scattered throughout its substance and definite areas of necrosis with increase in fibrous tissue was found.

The second case died and a partial necropsy exposing only the spleen and liver was done in 1919, the case being reported by Francis in 1927.

The third case was studied by Bruecken and was a complete autopsy with exhaustive study by fixed tissues, stained sections, cultures of heart blood and animal inoculations in 1926 and was reported by Francis in 1927. Circumscribed nodules were found which were both caseous and fibrous. The spleen was dark in color, quite soft and contained caseous nodules. The supra-renals contained opaque, yellow and grayish translucent areas. Lymph nodes were enlarged and showed yellow foci. The lungs contained enlarged peribronchial lymph nodes as well as old calcareous areas and fibrous tubercles. Enlarged lymph nodes were found in the omentum and near the ileo-cecal valve. The urine contained albumin, casts and pus cells. Cultures from blood obtained from the heart showed only streptococcus hemolyticus. Biopsy study of the lymph nodes found an increase in connective tissue fibers which contained nodules whose walls were made up of epithelioid cells interspersed with fibroblasts and the usual inflammatory reaction findings; the centers were necrotic, the blood vessel walls showed proliferating endothelial cells, a marked edema of the walls and reticulum increase.

The fourth case with the most complete pathologic study yet made was reported by W. Simpson from Dayton, Ohio. This was the most rapidly fatal case on record (four days, seven hours). The autopsy findings consisted of multiple focal necrosis in epitrochlear and axillary nodes, and in spleen, liver and right lung

In a study of pathological histology found in diseased animals by Strong and Councilman, they found miliary foci in the lymph nodes, liver and spleen of guinea pig which consisted of mononuclear cell accumulation, infiltration with polymorphonuclear leukocytes and necrotic areas. Necrotic areas were also found in the liver but no capillary lesion. Similar lesions were found in the adrenals, heart and lungs. There was also a general infection of the blood vessels, endothelial cells and the bacteria were

found in the vessels in all parts of the body. In the adrenal glands the cortical cells were the structures chiefly involved. The kidneys were infected throughout the glomerular structure and accumulation of mononuclear cells were found. They consider that the essential lesion is an infection of the endothelial cells, most marked in the blood vessels of such organs as the spleen and liver. They found staining of the bacterium in the tissues was difficult, though diagnosis could be definitely made in this way. Hemorrhagic areas in the lungs and kidneys were common.

The changes occurring in the vascular system take on the nature of an obliterative endarteritis most commonly. In the early lesions of the tissues, large numbers of histiocytes were uniformly present. In all the lesions of the cases which have been studied post mortem, the findings were sufficiently identical to be explained only on the basis of a common pathogenesis. As would be experienced in the pathological study of laboratory animals, those which are most highly susceptible show a more diffuse affection of structures. The white mouse has been found to lend the most ready demonstrations of the bacterium in smears and sections as well as in cultivating organisms from the blood. The lesions in the human show a rather marked contrast from those of susceptible animals, in that in the former, fibrous tissue proliferative cell changes are the most characteristic. This is explained on the basis of higher resistance and a degree of immunity. In the study of lesions in man, the similarity of appearance of tularemic changes to those incident to tuberculosis has often been emphasized. This confusion has been made more probable because Bâcerium tularense has not as yet been stained in sections from human lesions.

Immunity: Only one case of a second infection in man has been found in my study of the literature. This was reported by Lake and Francis and developed in a technician in the Hygienic Laboratory at Washington and developed two and one-half years after the first attack. It developed as a papule on the finger, was followed by lymphadenitis of the arm, presented no constitutional symptoms and the bacterium tularense was isolated by inoculation of a guinea pig. Since the agglutinins persist for many years in the blood of persons recovering from the disease the infrequency of second infections is easily understood. This degree of immunity appears not to obtain in susceptible animals, a circumstance not yet explained.

Diagnosis: It is frequently stated that tularemia is a new disease I think it would be more accurate to state that it is a newly recognized disease. From 1924 to 1926, the reported cases ran from fifteen to three hundred and twenty-three and the rec-thirty-four states. Up to May 1, 1928, 613 cases have come to the notice of Francis. Who believes that these changes in figures were the result of an actual increase in geographic distribution? Many observing physicians who now are recognizing tularemia are also recognizing identical cases seen before the time of its clinical identification but which they called

something else then. The rodents now recognized as the "reservoirs of infection," a phrase of Francis and Callender, have not made the United States their recent habitation. Rabbits and ground squirrels are indigenous. Neither have flies, fleas and ticks come to torment them only with the advent of 1911.

Another evidence of the truth of this conviction was introduced by the establishment of the identity of "Ohara's Disease" with tularemia. Ohara studied ân acute febrile disease in man contracted from handling wild rabbits in Japan. A number of cases fell under his personal observation and were rather carefully studied. He concluded that there was a causative relationship between the handling of uncooked rabbits and the illness of his patients. He further concluded that eating the cooked meat did not transmit the disease. The symptoms described in his cases were sufficiently identical with those recognized as tularemia in this country that further laboratory study including agglutination test of patients was carried out. Blood serum from seven of his cases were sent to the Hygienic Laboratory U. S. Public Health Service during the years of 1925 and 1926, they all agglutinated Bacterium tularense. Inoculation of guinea pigs was also done at the laboratory from human lymph glands removed from Dr. Ohara's patient and the cultures obtained were identical morphologically and culturely with those studies obtained from this country. Animals inoculated with this culture died presenting the usual typical lesion of tularemia and the pathological study of the glands of these animals corresponded with the pathological findings of cases studied originating in the United States. Francis and Moore concluded that Ohara's Disease was identical with tularemia .

Laboratory: Agglutination of Bacterium tularense is considered equally dependable, diagnostically, as isolation of the organism. Francis says, "I know of no other disease in which an agglutination test will set the diagnosis right in such uncertain terms after so many years."

The serum of the blood from the suspected case is obtained in the same way as serum for a typhoid agglutination test and is performed in a similar manner to the well known Widal. The blood should be obtained under sterile conditions and no antiseptic should be added. If the specimen is to be transported over a distance to the laboratory, pure glycerine may be added to the serum in equal amounts.

Agglutinins develop in the blood of the patient by the twelfth to fourteenth day of the disease. The degree of development may reach a dilution of 1-1200 or even more. The blood reaches a maximum agglutinin titer by the fourth or fifth week followed usually, by a gradual decline, though in one Lister Institute case the maximum was still noted at the end of 403 days. The agglutinin property lasts, probably, through life. The longest period of persistence was reported by Brosius where a case now thought to have had tularemia infection in 1909 was found by Francis to agglutinate Bacterium tularense in January 1928.

The germ is small, round or rod-shaped, 0.3 to 0.7 micron in length, 0.2 to 0.3 micron in width and is often capsulated. It stains poorly with methylene blue but fairly satisfactorily with carbol-fuchsin or gentian violet or Giemsa's solution. They are found in many organs of the body and in the blood. Pus from the initial ulcer, a suppurating gland or the ophthalmic secretions should be ground in a mortar, suspended in normal saline, strained and injected subcutaneously into the abdomen of a rabbit or guinea pig. If tularense bacterium is present, the animal should, within two weeks, show at post mortem, caseation of the inguinal glands and multiple foci of necrotic areas in various organs, particularly the liver and spleen.

Cultures may be obtained from the blood, liver or spleen of these animals. The media most satisfactory for culture growth are egg yolk and serum glucose cystin agar. No growth occurs on plain agar. A diagnosis from smears or culture inoculations cannot be made. The gross pathology found in the inoculated animal is of much more value diagnostically. Injection into rabbits or guinea pigs of blood from severe cases in man results in infection and death, a fact which establishes the bacteremic nature of the disease. Attempts to cultivate the organism from the blood of human cases have failed in most of the cases. Francis was able to recover it in two very severe cases. How long the germ lives after drying and exposure to air or sunshine has not been determined but it remains virulent for at least a month in spleens or livers of animals that have died from the infection, if placed in glycerine and kept in the ice box.

Clinical: In man the disease appears chiefly as a glandular and a typhoidal type. In the ulcero-glandular form the initial sore can usually be demonstrated. It takes on the nature of an ulcer (Fig. 1), sluggish in its appearance and process of development and healing (Fig. 2). They rarely attain great size, one to two

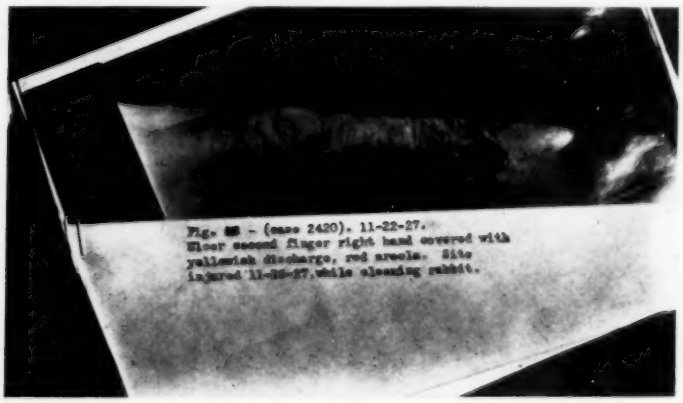
Fig. 88 - (case 2420). 11-22-27.
Ulcer second finger right hand covered with
yellowish discharge, red areola. Site
injured 11-20-27, while cleaning rabbit.

Fig. 1

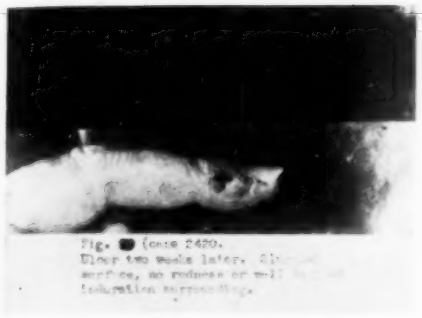

Fig. [redacted] (case 2420. Ulcer two weeks later. Slight surface, no redness or infiltration surrounding.

Fig. II

c.m. being the average diameter, are shallow, rather clearly defined with little or no redness or infiltration of surrounding tissue. They are superficial, 0.1 to 0.2 c.m. deep and the surface is covered with a grayish membrane not easily removed, hence but little discharge. The ulcer is rarely multiple (Fig. 6), but multiplicity seems to have no effect on the degree of severity of the other symptoms. It is usually painful but never assumes the degree of discomfort which usually accompanies an ordinary septic wound. The glands located in the drainage area generally swell, varying widely in size, are firm, fairly fixed and frequently become necrotic and require removal or drainage. The overlying skin may become reddened resembling the usual streptococci infection and the lymphatic vessels may become involved presenting a brawny feel to the examining finger. Usually the adenitis is confined to the extremity presenting the initial lesion but enlargement of the cervical glands and those of the opposite side of the body often occur. The glands may suppurate but this has not been common in our series. Those not suppurating may remain enlarged for several months.

I see no reason for not including the ophthalmo-glandular group of cases under the heading, ulcero-glandular type. Here the primary lesion is on the conjunctivae instead of the skin and constitutes the initial lesion which is both external and demonstrable. Conjunctival cultures are positive in such cases. The incubation period is short—three to five days being the average; two days is the shortest period and ten being the longest in the reported cases studied. The symptoms occur rather abruptly and consist of chilliness, myalgia, headache, prostration and fever. Pain is usually experienced in the swollen lymph glands within forty-eight hours after the prodromal symptoms and twenty-four hours before attention is attracted to the local lesion. The latter begins as a papule, rapidly necroses and is characterized by its sluggishness, three or four weeks often being required for healing.

The temperature ranges from 99 to 104, more frequently the maximum does not exceed 102. It is irregular in curve, resembling the course of a septic case and reaches normal in from two to three weeks. (Fig. 3.)

The blood picture is not characteristic. Leucocytosis of moderate degree occurs; if a high count appears it should suggest a mixed infection. The stained cell study affords no diagnostic help.

Judging from the post mortem findings in the liver of animals dying from natural or inoculated infections, one would expect jaundice as a rather frequent symptom. Verbrycke reported a

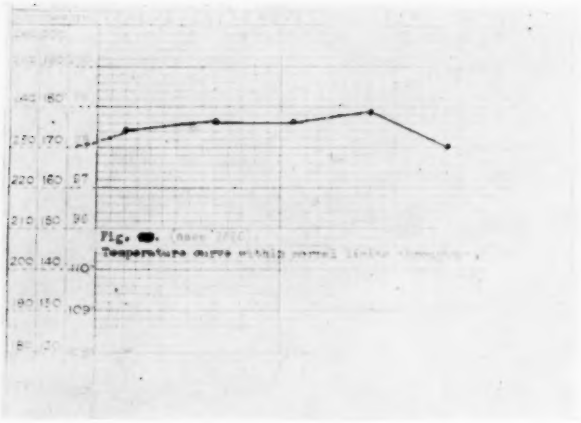

FIG. III

case with icterus which was subsequently necropsied. An additional feature of interest in this case was the sequence of events as learned by Francis in his study of the case; two days after dressing rabbits, the patient injured her right index finger with a splinter, the disease developing three days later. Fulmer reported a series of eighteen cases from Arkansas in which there was one death. This patient died on the twelfth day of illness with what was diagnosed as general peritonitis. No other cases of peritoneal involvement have been found to be recorded.

The other type resembles typhoid fever and runs a similar temperature range (100-104), but the remissions are usually accompanied by free perspiration. In this respect it resembles so-called septic conditions. Chills, likewise, are not uncommon in this type just as they occur in the ulcero-glandular type Prostration is quite marked even from the onset, persisting after the fever has passed. While the definite febrile period may be a bit shorter than in typhoid, complete convalescence is tardier; the keen desire for food and rapid gain in weight, rather the rule in typhoid, is absent in tularemia of the typhoid type. Another differential feature, clinically, is the absence of the rather frequent complications such as intestinal hemorrhage during the febrile period and phlebitis in the convalescing period. While the majority of the cases of typhoid type do not present glandular enlargements, it does occur. Two of the three cases developing in the Lister Institute had some gland involvement. The symptoms of such cases as do not show glandular enlargement are materially identical with the ulcero-glandular type with the sole exception of the primary ulcer and the gland swelling. In fact, the closer one studies the symptoms of one's own and reported cases, the more the conviction grows that they are, after all, tularemia; that symptoms in either type are merely suggestive and a diagnosis must rest on the serum reaction; and that a better classification

would be *tularemia with primary lesion and gland involvement* and *tularemia without demonstrable primary lesion and gland involvement.* Most of the reported cases of typhoid type have been in the person of laboratory workers. Kavanaugh's case was a housewife with a history of having handled a rabbit from which her daughter-in-law contracted the ulceroglandular type of the disease; the blood serum of this case agglutinated bacterium tularense in dilutions from 1:10 to 1:160 and terminated fatally.

No satisfactory explanation has yet been offered concerning the means of entrance of the germ in these non-ulcerative, non-glandular cases. Conveyance of the infection from the hands to the mouth to be swallowed must be considered a possibility. Whether the germ may be inhaled as occurs with the tubercle bacillus is considered as a possibility when one has seen the post mortem pulmonary lesions in animals. Francis suggests the possibility of the entrance through unbroken skin surfaces because of the presence of cystin in the skin and the known cystin requirement in media for the organisms growth. Just why a papule should not be formed when the organism is absorbed through unbroken skin and yet colonize in a broken area with ulcer formation and gland involvement is not clear.

Walter Simpson has reported a fatal case of only one hundred and three hours duration and quotes a letter from Francis relating three cases in one family of short duration, death occurring on the 6th, 8th and 9th days respectively. The occurrence of 49 cases in his series observed in Dayton in four months would seem to indicate a more common incidence of the disease than hitherto appeared. He was able to trace twenty-five additional cases occurring prior to 1927 dating back to 1908. On the other hand, in the county of Harlan, Kentucky, where this paper was read, a country where ticks and wild rabbits abound, no cases had been recognized by more than twenty-five physicians present.

Convalescence in the man is the rule, indeed death may be said to be rare. Recovery is, however, very slow; three or four months after the infection may still find the patient a semi-invalid, but when it does occur it appears to be complete. I have seen no deaths but three months incapacity was observed in one case and five in another. This latter case merits some detail.

Case 3140-S. A farmer, age 37, was seen in consultation with Dr. Rush, Fern Creek, Kentucky, December 8, 1925. He was first seen by Dr. Rush about November 7, 1925, on account of an infected finger caused by a table fork while eating a rabbit which he had just cleaned. He was put to bed and one week later began to have pain in the precordial region and dyspnea. He ran a temperature from 101 to 103, perspired freely and had profuse night sweats. There was some cough and a blood-tinged sputum was observed for several days. There was marked prostration. He had enjoyed lifelong good health and his family history was negative except for one brother who had died of pulmonary tuberculosis.

Examination, physically, showed a well developed and well nourished man with the appearance of being acutely ill; respiration was labored, feeble respiratory sounds and a modified percussion note were noted over the lower lobe of the left lung, outer half, posteriorly, while over the inner half ex-

tending two inches from the spine was an area of flatness and bronchial breathing. The apex beat was indistinct but the point of greatest intensity was inside the dull area. A friction rub was heard to left of sternum over the fourth intercostal space and scattered moist râles over the left lung root. Capillary circulation fair and an increased cervical pulsation was noted.

Blood pressure:	
Systolic	105
Diastolic	62
Heart Rate	86
Pulse Pressure	83
Mean Pressure	43
Heart Load	69%

The ulcer on the right index finger was two cm. in diameter, with irregular edges, filled with a purulent discharge and surrounded by a narrow zone of redness and induration. The olecranon and axillary lymph nodes were enlarged, firm, movable and tender on the right side, while on the left side the axillary glands were enlarged but smaller.

12-8-25

Blood Count.	
Red Cells	5,440,000
White Cells	10,350
Hemoglobin	90%
Color Index	.86
Stained Specimen:	
Polys.	72%
Mono. L.	7%
Mono. S.	19%
Eosin.	2%

12-8-25

Urinalysis.	
Appearance	Straw color.
Reaction	Acid
Sp. Gr.	1023
Albumin	Trace
Sugar	0
Acetone	0
Diacetic	0
Indican	0
Microscopic:	
Occasional Red Blood Cell.	
Urates.	
Occasional hyaline cast.	

12-9-25

X-Ray Study of the Chest:

Fluoroscopic examination of the chest shows a greatly dilated cardiac shadow, the left margin of the heart comes within one inch of the left lateral chest wall. The dilatation to the right side is three inches to the right of the spine. The aortic and great blood vessel shadows do not appear increased in size. After the patient's head had been elevated fifteen degrees, there was no apparent change in the contour of the cardiac shadow. No examination was made in the erect posture.

On the antero-posterior film, made at a 36 inch distance in the supine position shows the cardiac shadow as revealed in fluoroscopic examination. The transverse measurement at the base is ten inches. There is a general mottling of both lung fields which is probably due to passive congestion. It appears to be very symmetrical in outline and appears to involve all portions of the lungs. (Fig. 4.)

12-10-25

Urinalysis.	
Reaction	Acid
Sp. Gr.	1022
Albumin	3 plus
Sugar	0.25%
Acetone	0
Diacetic	0
Indican	Trace
Microscopic:	
Squamous & Renal Epith. Cells.	
Red Blood Cells, many.	
Oxalate calcium crystals, few.	
Occasional granular and hyaline cast.	

Aspiration of pericardial fluid was done for diagnostic reasons, and 30 cc. of fluid removed.

Laboratory Examination:

Straw colored.
Slightly cloudy.
Red Blood cells few.
Lymphocytes many.
Short rod shaped bacilli.
Few streptococci.

Temperature chart, graphic.
Fig. 5.

FIG. IV Case No. 3140-s. X-ray of chest, antero-posterier, 36 inches, Recumbent.

Blood serum agglutinated bacterium tularense to 1-60 dilutions.

The wound of index finger slowly healed. Smears from pus showed staphylococci and streptococci.

Discharge from Hospital on own request, improved, January 6, 1926.

On March 11, 1926, he was again examined, looks fairly well. Valve sounds negative. Apex beat inside mammary line. Heart regular and rhythmic.

Blood pressure:

S.	132	P.P.	122
D.	112	M.P.	20
H.R.	85 sitting	H.I.	26%
	144 erect.		

Lungs clear. Lymph glands in right axilla still large, firm, movable and tender. Submaxillary gland on right side enlarged, not tender.

Blood Count.	
Hemoglobin	85%
Red Cells	4,560,000
White Cells	8,300
Polys.	69%
Lymphs. L.	7%
Lymphs. S.	20%
Eosin.	2%
Baso.	2%
Urinalysis.	
Reaction	Acid.
Sp. Gr.	1030
Albumin	0
Sugar	0
Acetone	0
Diacetic	0
Bile	0
Microscopic:	
Occasional pus cell.	
Urates.	

10-18-26

Weight 190 lbs.

Heart rate 84. Valve sounds negative. Chest clear. No friction rub. Complained some discomfort on exercise in precordial region.

Case 2826. Is reported because of two features—multiple ulcers and absence of lymph node enlargement.

Female age 43, was seen on account of ulcerating wounds on the thumb and little finger of the right hand and moderate pros-

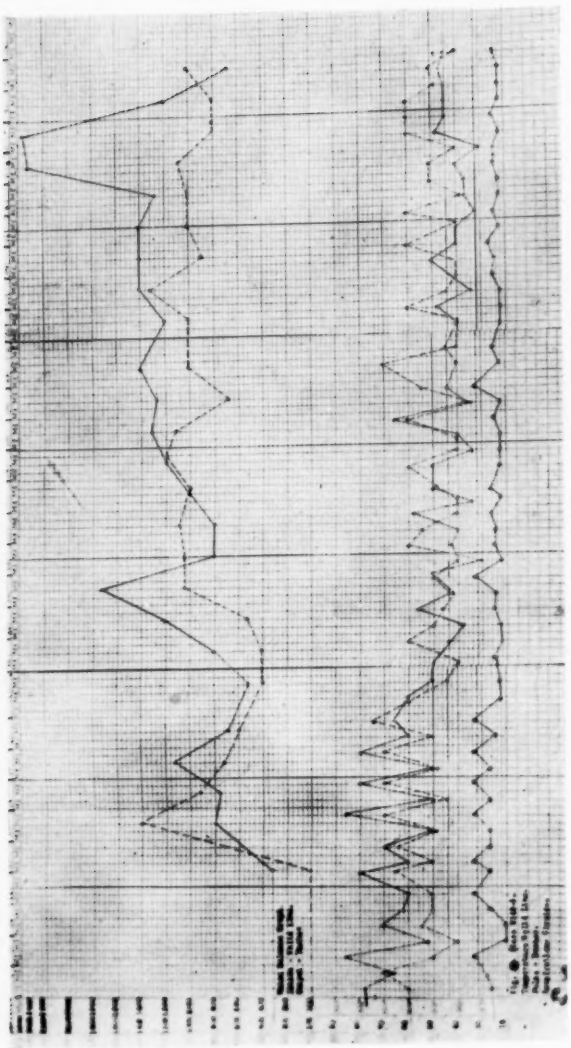

Fig. V Case No. 3140 s. Temperature chart from 2-8-25. To 10-5-26, when he left hospital on own request.

tration. The ulcers were small and showed but little discharge. (Fig. 6.) The glands of the right arm and axillary space were apparently normal in size and not tender. The temperature was normal and remained so at time of each visit. (Fig. 7.)

12-5-27

Blood Count.	
White Cells	7,400
Hemoglobin	85%
Polys.	62%
Small Lymphs.	29%
Large Lymphs.	9%
Eosin.	0
Baso.	0

12-7-27

White Cells	9,600

12-22-27

White Cells	11,030

FIG. VI Case 2826 multiple ulcers; photographed ten days after development.

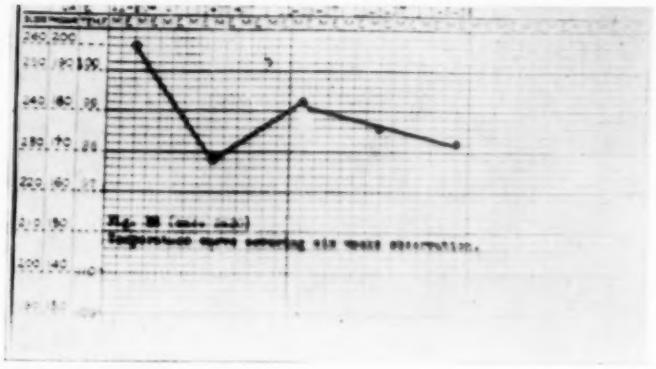

FIG. VII Temperature Curve, same case, from 12-2-27-to-1-10-28.

1-25-28	
White Cells	9,500
Hemoglobin	75%
Polys.	44%
Small Lymphs.	48%
Large Lymphs.	3%
Eosin.	1%
Mononuclears	4%

Blood serum agglutinated bacterium tularense in dilutions 1-180.

Summary: The unusual evolution of our present knowledge of the disease is traced.

Attention is called to the high degree of transmissibility of the disease to laboratory workers and the relative rarity of the disease through ordinary modes of transmission.

The high fatality to animals contracting the disease either through inoculation or by natural means and the low incidence of death in man is stressed.

It is suggested that the nomenclature be changed.

Tularemia with initial lesion and glandular involvement and tularemia without demonstrable initial lesion and glandular involvement being considered more comprehensive than the present classification.

The rarity of man to man inoculation is considered.

The apparent life-long immunity of the disease in recovered cases in man is emphasized.

A case with pericardial effusion with recovery is reported.

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Editorial

MARCELLO MALPIGHI
1628-1694

It was a rather striking and interesting coincidence that the anniversaries of three men to whom we owe so much of our knowledge concerning the vascular system should have been celebrated in 1928. William Harvey's great masterpiece "De Motu Cordis et Sanguinis" was published in 1628 at Frankfort by an obscure German publisher, Wilhelm Fitzer; in the same year Marcello Malpighi was born on a little farm at Crevalcuore near Bologna; and in 1728 John Hunter came into the world at the village of Long Calderwood, Lanarkshire. Truly three of the greatest in the history of medical science, to whom the medical and scientific world paid due honors in 1928! It was especially fitting that Malpighi's anniversary should have been celebrated at the same time with that of Harvey, for it was he, who in his first published work in 1661, supplied the missing link in Harvey's chain of investigations—that of the capillary circulation. And this was made possible through Malpighi's use of the microscope which Harvey did not possess and consequently could not see the capillaries. It has been well said that Malpighi made a histologic certainty of what Harvey had made a logical necessity. About 1650 there began to be constructed and employed in scientific investigations crude microscopes employing simple lenses of high power; and with such instruments many of the most important discoveries of the second half of the seventeenth century were made. With such a lens Malpighi saw for the first time the capillaries in the lung of the frog. Luck was with him in his selection of an organ for this research, because of the transparency of the frog's lung and its relatively conspicuous capillaries. At any rate he saw them and recognized their function for he describes in the rare first edition of his work "De Pulmonibus," Bologna, 1661, the passage of blood from the arteries to the veins through the "capillary" blood vessels. Harvey had only inferred their existence, Malpighi demonstrated it. In a figure in this work the capillary network of the wall of the pulmonary sac is clearly indicated, as is also the vesicular nature of the lung tissue. He also saw a similar capillary network in the wall of the frog's distended urinary bladder. This important discovery was recognized by the Royal Society of London; and it was republished at Leyden and in other places in the years following. This discovery may well be considered the beginning of microscopic anatomy; and Malpighi may be considered its founder, so important were the discoveries made by him with his simple microscope. He applied it to the

study of both animal and vegetable structure. At a very early period he concerned himself with the study of plant histology in anticipation of the more difficult investigations on the histology of animal tissues. He had knowledge of the spiral vessels of plants as early as 1662, but it was not until 1671 that he wrote his "*Anatome plantarum.*" This he sent to the Royal Society, who published it in the next year. Just a few months before its publication there had been brought out in London under the title of the "Anatomy of Vegetables" an English work, by Nehemiah Grew, which clouded considerably Malpighi's priority as a plant histologist. However, his priority in animal histology along so many important lines is so incontestable that the question of his priority in plant histology becomes a matter of very secondary importance. In the Anatome plantarum there was included an appendix, "*Observations de ovo incubato*" which described the development of the chick, particularly the later stages, surpassing in many points of detail the work of Harvey along the same line. Unfortunately he developed the metaphysical conception of predelineation in the undeveloped ovum in connection with his objective observations, to the impairment of the value of the latter. Harvey's work on "Exercitationes de Generatione Animalium" had appeared in 1651, twenty years before. Malpighi, applying his microscope, was able to describe in detail the development of the organs of the embryo, particularly of the heart and nervous system. He thus laid the foundations of microscopic embryology. But the researches of Malpighi on the structure of the brain, liver, spleen and kidneys were perhaps of as great importance as his discovery of the capillaries, equally epoch-making in character. In an epistle addressed to his old fellow student, Fracassato, *De cerebro exercitatio epistolica,* he showed that microscopically the white matter consisted of round but flattened little fibers arranged in bundles forming tracts connecting the surface of the brain with various regions of the spinal cord; further he showed that the gray matter is not confined to the surface of the brain where it is called cortex, but exists in scattered masses in the interior, disposed around the ventricles and along the cord. In this epistle he was unwilling to define exactly the nature of the gray matter, but later, influenced by his work on glands, he came to the conclusion that it was of a glandular nature, describing the superficial gray matter as "glands hanging on to the strands of the white fibers like the fruit of the date palm." At the same time he was working on the tongue and skin, discovering in the latter the lower layer of the epidermis which we still call after him the rete Malpighi layer. In 1666 he published the results of his investigations on the glandular organs under the title of *De viscerum structura exercitatio anatomica.* He cleared up the mystery which had so long hung over the liver by showing that it was constructed after the fashion of a conglomerate secreting gland, that its substance was arranged in small masses essentially like those of which other conglomerate glands such as the pancreas and salivary glands

were made up, and to these he gave the name of *acini*. He regarded the common duct as the excretory duct of the liver, the liver secreting bile as the salivary glands secreted saliva. He did not pretend to answer the questions by "what mechanism is the bile separated in the glandular acini of the liver and what is the use of bile in the economy," but expressed himself as "contented to have described the simple and rude structure of the liver." Nevertheless Malpighi's description of the microscopical structure of the liver remained with little change up to the present century. Similarly Malpighi came to a clear conception of the microscopic structure of the kidney. He showed that in man the kidney was really made up of several kidneys consisting of masses of tubules arranged in the form of pyramids, since known as the pyramids of Malpighi. He showed that the tubules constituting these pyramids ended in orifices at the summit of the papilla which formed the apex of the pyramid, and further that in the cortex of the kidney the tubules were not straight but irregularly twisted. He showed further that these tubules begin as inflated swellings or capsules, "round like the eggs of fishes," containing a knot of blood vessels, hung on to the small arteries as "apples hang on to the branches of a tree." The Malpighian tufts or corpuscles we have called them ever since. Malpighi was convinced that they played an important part in the secretion of urine. Practically our knowledge of the kidney structure remained where Malpighi left it until the time of Bowman. Finally the nature of the structure of the spleen was revealed through Malpighi's observations. He gave a thorough description of its capsule, trabeculae, pulp, blood vesesls and nerves, showing it to be not a gland but a contractile vascular organ. He was the first to observe the small white bodies, not unlike glands, attached to the blood vessels, which ever since have been known as the Malpighian corpuscles. During the remainder of his life Malpighi continued to work on anatomical and physiological problems and wrote important tracts on the uterus, hairs, horns, bone, cardiac polypi, the lymphatic glands, anatomy of plants, natural history of the silk-worm, but none of these attained results equal to the epoch-making investigations already mentioned. His writings on the nature and causes of disease justify for him the credit of being regarded as having laid the foundations of scientific pathology. Malpighi's name stands out as the fore-runner of modern medical science. He represents the beginning of a new epoch, that of the microscope. He is truly the father of microscopic histology and embryology. It has been said of him that he left his mark upon whatever branch of natural history he touched. He may be called a zoologist, a structural botanist, a comparative anatomist, an embryologist, histologist, pathologist, physiologist or biologist. He stands out as the first researcher who comprehended the essential identity of plant and animal life. Upon every problem that he touched he shed light. Malpighi's personality has been described as most attractive; he was gentle, patient, fair-minded, sensitive

and sympathetic, and a devoted practitioner of medicine. In study he was accurate and untiring, exceedingly bright and quick of comprehension, and moved to the depths of his being by a passion for the new learning. He was delicate in constitution and subject to frequent illness. His life was uneventful, and without romance. He was honored throughout the world and beloved at home, he had many scientific friends and correspondents, but peaceful in disposition as he was, his long life was made unhappy from time to time by attacks made upon him by a neighboring family with whom there existed a life-long feud arising out of disputes over property boundaries. He was most happily married but was childless. He entered the University of Bologna in 1645 as a student in philosophy, but as the result of the deaths of his father and mother, family cares, and the aforementioned feud, his studies were interrupted until 1651, when he again definitely took up his studies with the intention of studying medicine. In 1653 he became a Doctor in Medicine and Philosophy, and after three years of medical practice was made a Professor in Medicine. In the same year there was created for him a new special chair of Theoretical Medicine at the University of Pisa which he held for three years, teaching and experimenting. Because of domestic difficulties in Bologna, and the unfavorable climate of Pisa he resigned his chair there and returned to Bologna where he spent the next four years in teaching, practice and research, developing the ideas that later were to come to such splendid fruition. In 1662 he was offered the chief chair of Medicine in the University of Messina which he held for four years. Pressed to remain longer he asked permission to make a visit to his native city. Received most warmly in Bologna as a token of the great fame his researches had already brought him, he was offered the Chair of Medicine in his home city. Released by the Senate of Messina, much against their desire, Malpighi once again took a chair in his old University, which he held for a quarter of a century, lecturing, practising medicine and giving the best energies of his mind to his investigations. In 1667 his connection with the Royal Society of London was begun, which led to a long and close intercourse between Malpighi and the English Society. As a result of this the Royal Society had the honor of publishing the greater part of Malpighi's works which he produced after his return to Bologna. The years passed uneventfully; he spent his winters in the city busy with teaching and practice, his summers in the country in rest and research. The loss of many of his manuscripts through a fire in his house in Bologna in 1684 left him much broken; and the old family feud continued to harass him, almost to his last days. In 1691, Pope Innocent XII invited him to come to Rome as the Pope's physician. At first refusing, Malpighi was at last unwillingly made to accept the invitation. Soon after his arrival there he became ill; had a slight apoplectic stroke in 1694, and another more severe a few months later from which he died on the 29th of November,

1694. Thus passed a very great man, the one who most truly marks the beginning of modern medicine. His name is eponymically preserved for us in the rete Malpighi, the pyramids of Malpighi, the Malpighian capsules of the kidney and the Malpighian follicles of the spleen. Would that every medical student as he comes into contact with this name might be inspired to read these epoch-making works which these terms commemorate!

Abstracts

Heat Stroke a Thermoregulatory Incompetency. By EBEN E. SMITH, Lt. Commander, Medical Corps, U. S. N. (U. S. N. Med. Bull., Vol. 26, No. 5, 1928).

The present discussion is concerned with those phenomena resulting from failure of a normal individual to maintain normal physiology under influences favorable to heat accumulation. Smith draws the following conclusions: Heat stroke is a thermoregulatory incompetency resulting from exposure to heat. The clinical manifestations of heat stroke are of several types, but these various conditions have a common pathogenesis. Thermoregulatory failure may occur through cardiovascular collapse before hyperthermia develops. Hyperthermia develops through failure to dissipate heat. It results from an unbalance of reaction between complex physical and physiological factors. Profound acute metabolic disturbances occur in heat stroke. The trauma resulting from heat stroke affects parenchymatous tissue and may cause a fatal morphological disorganization. Tetany is an important element in heat stroke. It commonly affects the heart and may cause sudden death. It also affects smooth muscle and may prevent water assimilation through intestinal spasm. Heat, per se, is not the cause of the tissue trauma. The response to heat exposure develops toxic factors when thermoregulation fails. The nature of these factors is hypothetical and undetermined. Loss of water and salt is a significant fact in that it is a very important element in causing thermoregulatory failure and cramps. It probably also seriously embarrasses metabolic processes. An uncompensated acidosis occurs in heat stroke. This, as yet, can be attributed to no single acid body, and the condition probably results from the accumulation of the various acid products of metabolism. An acute interstitial pneumonitis occurs. It is of sufficient degree to decrease materially the vital capacity and retard respiratory exchange. Summing up our known knowledge of the condition of thermoregulatory failure, heat, accelerated metabolism, depletion of tissue fluid, decreased respiratory exchange, increased hydrogen concentration, and parenchymatous degeneration represent the sequence of essential events in the pathogenesis of heat stroke.

Effects of Communicable Diseases on Thyroid Gland. By ROBERT OLESEN, Surgeon, United States Public Health Service (Public Health Reports, November 16, 1928, No. 46).

Infections and intoxications have long been believed to constitute etiological factors in the production of goiter. Many English observers, notably McGarrison, have relegated iodine to a minor etiological rôle. He considers goiter to be an infectious waterborne disease, the exciting cause of which is an organism belonging to the colon group. Crotti also strongly supports the infectious origin of simple goiter, holding that it is due to an organism of the trypanosoma type. Most Americans regard simple goiter as a deficiency disease. Investigations by Olesen and Taylor among school children in Cincinnati, Ohio, and the State of Connecticut failed to show any relationship between diseased tonsils and decayed teeth and simple goiter. Studies by Hertzler and Bram likewise failed to establish such a connection. The present article treats first of the thyroid status of 589 boys and 636 girls who were examined before and after they had had certain communicable diseases. In the accompanying control group the thyroids of 1842 boys and 1783 girls who did not have communicable diseases during the interval were examined one year apart. After the illness no changes in thyroid size were detectable in 51.7 per cent of the boys and 45.6 per cent of the girls. Thyroid

size was diminished in 13.8 per cent of the boys and 11 per cent of the girls. Increased size was noted among 34.5 per cent of the boys and 43.4 per cent of the girls. The thyroid of 1842 boys and 1783 girls who did not have communicable illness in the interval were examined one year apart. No change in thyroid size had taken place among 48.4 per cent of the boys and 43.6 per cent of the girls. Thyroid size had decreased in 12.7 per cent of the boys and 18.6 per cent of the girls, while an increase was recorded among 38.8 per cent of the boys and 35.7 per cent of the girls. A communicable disease census of 7,977 boys and 8,441 girls showed little difference in percentage incidence among the thyroid-normal and the thyroid-enlarged children who had the same maladies. There is some evidence to show that one of the immediate effects of communicable diseases among girls of elementary school age is a simple enlargement of the thyroid gland. However, this thyroid enlargement appears to be temporary in character. A comparatively short time, the length of which is as yet undetermined, after a child recovers from a communicable disease, he is no more prone to changes in thyroid size than a child who has not had a communicable disease. In so far as elementary school children are concerned, there appears to be no ground for assuming that the ordinary communicable diseases are responsible for goiter. The underlying causes of this malady must be sought for in other directions.

Liver Diet and Tar Cancer. By J. Maisin and A. Francois (Annales de Médicine, Paris, 24, 1928, p. 345).

Two groups of mice were employed for this investigation; controls, which were fed with wheat flour, corn and white bread, and the second group that received in addition to this same diet, 5 gm. of fresh liver three times weekly. All of the mice of both groups were painted with tar three times a week for 120 days. On the one hundredth and twentieth day cancer was observed in 21.5 per cent of the mice kept on the liver diet, but only 4.16 per cent of the controls not fed with liver developed cancer. On the two hundreth and tenth day cancer was observed in all mice fed on the liver diet, but only 62.86 per cent of the controls had developed cancer. The percentages of pulmonary and axillary metastases were 37.9 and 59.4 respectively in the controls, but were 55 and 72.6 in the liver-fed mice. The control mice lived seventy days longer on the average than did the mice fed with liver. The same results were obtained when dried and ether washed powdered liver was substituted for the fresh liver in the diet. These results appeared to the investigators as highly significant; they explain the situation by the assumption that the liver acts in these cases, not simply by increasing the protein content of the diet, but by means of some specific substance similar to that acting upon the bone-marrow in pernicious anemia. If these experiments of Maisin and François can be confirmed and prove to be constant results of liver feeding in the production of tar-cancer, it will prove to be a discovery of first class importance in cancer research.

Die Wirkung des Insulins auf die Lipochromämie und die Xanthosis Diabetica. By A. Wl. Elmer and M. Scheps (Klinische Wochenschr., February 12, 1929, p. 300).

The previous investigations concerning xanthosis diabetica have not considered the effects of insulin upon the lipochromemia and its clinical changes. Elmer and Scheps have had recent opportunity of studying five cases of xanthosis diabetica, and the effect of insulin upon the lipochromemia and the skin coloration. These observations showed that the lipochromemia is dependent upon the nature of the food given to the diabetic, such as eggs, spinach, carrots, etc., that are rich in lipochrome, and that xanthosis diabetica appears in some cases shortly after such a diet is initiated, and slowly retrogresses when such lipochrome-rich diet is stopped. This retrogression of the xanthosis takes place more quickly when insulin is given than when it is not used. When a lipochrome-rich diet is used insulin cannot prevent the formation of xanthosis diabetica, yet it acts so that the lipochromemia does not reach such a high degree as it would in the absence of insulin, and the

skin coloration is correspondingly less. In three other cases in which xanthosis diabetica had appeared after a lipochrome-rich diet, it was possibly by the use of insulin to lessen the degree of skin coloration, but without restricting the lipochrome diet neither the appearance of xanthosis could be prevented nor its disappearance be brought about. These observers further showed that insulin affected the alimentary lipochromemia in diabetic cases without xanthosis. They conclude that insulin has an influence upon the lipochrome metabolism; in cases of xanthosis diabetica it produced a lowering of the lipochromemia, and prevents often the appearance of the skin coloration, or at least reduces it greatly. It increase the action of a lipochrome-free diet in that it hastens the disappearance of the xanthosis. In the diet-resistant cases of xanthosis diabetica it is recommended that insulin be used in aid of the lipochrome-poor diet. Insulin alone, without the help of such a diet, can in many cases not prevent the appearance of xanthosis diabetica. The significance of the action of insulin upon the lipochrome metabolism is very interesting, in that it would appear to indicate that in xanthosis diabetica the nutritive factor is not alone concerned, but that also a disturbance of lipochrome metabolism plays an important rôle.

Beiträge zur Lehre von den angeborenen Herzfehlern. By W. Redisch and H. Rösler (Wiener Arch. f. innere Med., 1929, Bd. VXI, p. 463).

Rösler has been engaged in the study of congenital heart lesions for the last three years. He has shown that the analysis of the röntgen findings has advanced essentially the differential diagnosis of these cases, much more than was to be expected from the existing literature upon the subject. Further, the study of his cases showed that consanguinity was an etiological factor of great importance, and perhaps alcohol. In continuing the systematic study of these cases the capillary microscope has been used, and the present paper deals with its application to the study of congenital heart lesions. Thirty-six cases were investigated by this means; in these particular attention was paid to the appearance of cyanosis, particularly of distal parts, to drumstick fingers and toes, and the corresponding capillary picture in the limbus of the finger- and toe-nails. For the purposes of comparison a second series of acquired heart disease of various kinds, nine cases in all, was studied. As a result of this study the authors conclude that there is a characteristic capillary picture for severe congenital lesions of the morbus coeruleus type. This capillary picture is dependent upon a capillary dilatation which is regarded as the cause of the morbus coeruleus. There is no connection between the capillary dilatation and the development of drumstick fingers. In isthmus stenosis of the aorta the capillary picture is characterized by numerous capillary aneurysms, and by the delicacy and smallness of the capillaries of the toes in contrast to the capillaries of the upper extremities. This finding occurred in the two cases of stenosis of the aorta examined, and was absent in all of the other forty-four cases studied.

Reviews

Recent Advances in Bacteriology and the Study of the Infections. By J. HENRY DIBLE, M.B. (Glasgow), M.R.C.P. Professor of Pathology and Bacteriology in the Welsh National School; Late Professor of Pathology in the University of London. 363 pages, 22 illustrations. B. Blakiston's Son & Co., Philadelphia, 1929. Price in cloth: $3.50.

This is an attempt by a general student and reader of medical bacteriology to present in a readable form some of the more recent changes in the subject and to indicate the lines upon which it is evolving. It has been the author's endeavor to take a broad view of many subjects, and, in keeping the balance between extreme technicality and what is already common knowledge, to present a readable exposition of recent work which the general medical reader may appreciate. At the same time the author seeks to indicate to those who do possess some knowledge of bacteriology and the infections what is being done in spheres outside of their own. The rapid evolution of the subject makes it impossible to take notice of all of the most recent work. The period covered is, therefore roughly that from the commencement of the War to the beginning of 1928. The writer has, perforce, set out a good deal of matter upon which at the present time judgment is suspended, and views which are undoubtedly speculative. He disclaims all responsibility for the accuracy of quoted work at times, and where it has seemed desirable, he has endeavored to place before his readers a judicial summing up of most questions. There are seventeen chapters and an Index. The first deals with the classification of bacteria. This is based upon the nomenclature adopted by the Society of American Bacteriologists and extended by Bergey in his textbook published in second edition in 1926, which represents a serious effort to unify bacterial classification and to combine the points of view of the general and medical bacteriologists. In this classification the International Rules of Botanical Nomenclature have been adopted in so far as they can be made to apply to bacteriology. The second chapter considers the streptococcus problem as shown in scarlet fever, erysipelas, puerperal sepsis and acute rheumatism. Chapter three treats of bacterial variation, chapter four of the bacteriophage, and five of experimental epidemiology. The remaining chapters treat successively of Calmette and B. C. G., ultramicroscopic and filter-passing viruses, diseases asociated with Rickettsia bodies, measles and tularemia, recent work upon the pneumococci, spirochetal infections, local immunity and the work of Besredka, diphtheria and the anaerobic organisms. The most important work bearing upon the various subjects treated is well analyzed and digested, the discussion is fair and dispassionate, and the conclusions conservative and well-guarded. There seem to be some important omissions, for instance, the important work of Mellon on transmutation is not mentioned in the chapter on bacterial variation. The work of the last four years is not as thoroughly sifted as that of the war period and immediately following. This is a thoroughly useful little resumé of the recent advances in bacteriology, and very convenient for the general reader and for the student and teacher. The author's style is especially well adapted to a critical review of this kind, being clear and concise, and this added to his unprejudiced treatment, makes of it a very useful book.

A Handbook of Clinical Pathology. By FRANK SCOTT FOWWEATHER, M.D., M.Sc., D.P.H. (Liverpool), F.I.C., Lecturer in

Chemical Pathology, University of Leeds, Chemical Pathologist, Leeds General Infirmary. With Foreword by Sir BERKELEY, Bt., K.C.M.G., C.B., President of the Royal College of Surgeons of England. 216 pages, 18 illustrations. P. Blakiston's Son & Co., Philadelphia, 1929. Price in cloth: $3.00.

This little volume is based on a course of lectures on clinical chemical pathology given to senior students of the Leeds University Medical School. In presenting them in this form it is hoped that they will serve as a short textbook on the subject which may be of use not only to medical students but to house surgeons and house physicians and to general practitioners. The book is not a handbook for laboratory workers since no attempt is made to describe methods of chemical analysis; nor is it intended to be a guide for research workers. It is intended primarily for the clinician, who should have a clear knowledge of the help he may expect from laboratory tests, of their limitations and of the conditions to be observed in order to get the best results from them. In other words, the clinician should know when to have the tests made, how to obtain the necessary specimens and how to interpret the results. It is with the object of offering information on these points that the book has primarily been written. Actual chemical work may be left in the hands of the expert. The book represents a fairly complete resumé of modern clinical chemistry along the lines indicated above. The material embraced in it is fairly complete, although some important omissions are noted. Taken all in all it fulfills the functions for which it was written very adequately, and may be recommended for this purpose. Its chief advantage is its simplicity of treatment.

Morphologic Variation and the Rate of Growth of Bacteria. Volume I of Microbiology Monographs: General. Agricultural. Industrial. By ARTHUR T. HENRICI, M.D., Professor of Bacteriology, University of Minnesota. 208 pages, 36 illustrations, 27 tables, 89 references. Charles C. Thomas, Springfield-Baltimore, 1928. Price in cloth: $3.00 net.

This is the first colume of a series of Monographs on General, Agricultural and Industrial Microbiology, edited by R. E. Buchanan of Iowa State College, E. B. Fred of Wisconsin and S. A. Waksman of Rutgers. These monographs will aim to present the subject of microbiology in the form of a series of monographs by authors of repute and authority in an authoritative and serviceable manner. These will treat of certain phases of microbiology in general or of personal investigations that can be treated in a monographic manner. The first volume is concerned with morphologic variation and the rate of growth of bacteria, one of the most important problems of the bacteriology of today. It is a record of personal researches undertaken with the hope that by the "magic of numbers" some order might be brought out of the chaos which has so far filled that field of bacteriology which has to deal with the form and structure of bacterial cells. The dogma of monomorphism has dominated the field of bacteriologic thought since the acceptance of the Cohn-Koch doctrine of the constancy of cell forms and the immutability of bacterial species, which has discouraged all investigation of problems of morphology, inheritance and variation in bacteria for a good many years. It is to be looked upon as a healthy sign that within recent years, through the activities of a small but persistent group of modern pleomorphists, bacteriology is at last definitely breaking away from the old tradition, and is seriously reopening for discussion and investigation the old problem of morphologic variation in bacteria. This movement has within it that danger inherent in all revolutionary movements, the tendency to go too far in the opposite direction. In the writings of at least some of the new pleomorphists, their protests to the contrary notwithstanding, there is clearly evident a tendency to return to the pre-Koch idea that any bacterium may transmute to the form of any other. It is high time, there-

fore, that we stop to evaluate the data and critically analyze the logic of the disciples of the "newer biology" of bacteria. The work of the latter investigators have probably not convinced more than a few that bacteria exhibit complex fungoid life cycles, but they demonstrated beyond all question that bacteria do regularly show pronounced morphologic variations, the nature and significance of which must be determined before we can make any real progress towards understanding the fundamental biologic problems of the group. In this work Henrici shows that bacterial cells are constantly changing in size and form and structure; but instead of these changes occurring in a haphazard or meaningless fashion, or instead of being phases of a rather vague and complex life cycle, they occur with great regularity, and are governed by relatively simple laws which, after more data have been accumulated and analyzed, may probably be very precisely formulated. Henrici's investigations indicate that the growth of bacteria in artificial cultures is governed by the same laws as govern the development of a multicellular organism; that their cells during growth pass through exactly the same sort of a developmental cycle as the cells of a plant or animal, exhibiting in turn an embryonic form during the period of slow growth or rest, and a senescent form during the period of death; that in short we may speak of a "cytomorphosis" in populations of free unicellular organisms differing only in degree from that of multicellular individuals. If this hypothesis be accepted it must prove of great importance in general biology, for it carries with it the implication that multicellular organisms are after all but populations of cells, whose organized individuality is a result, not a cause, of the differentiation of those cells. The morphologic variations of bacteria are therefore an expression of the variations in growth rate. The growth of a population of bacteria in a culture is apparently governed by the same laws that govern the growth of a multicellular individual; there is a close parallelism between the nature of the cell changes exhibited by a growing culture of bacteria and those which occur in a multicellular individual. If Henrici's views are correct, the monomorphistic theory without modification can no longer be held to be sound; but the theory of complex life cycles maintained by the pleomorphists remains to be proved by those who declare such life cycles to exist. The theory of cytomorphosis is not necessarily opposed to that of complex life cycles; rather the two viewpoints are rather different ways of looking at the same thing than that they are mutually exclusive. The ten chapters are concerned with: Problem of morphologic variation of bacteria; rate of growth of bacteria; technique; size of the cells of *Bac. megatherium;* size and form of the cells of the colon bacillus; observations on a diphtheroid bacillus; note on spore formation; morphologic variations of the cholera vibrio; senescent forms of the colon bacillus; and cytomorphosis in bacteria. Henrici's mongraph is a timely and valuable contribution to the vexed problem of bacterial variation. His work and logic seem very reasonable. It remains now for the pleomorphists to prove that his "age-forms" do actually represent stages in a complex sexual cycle.

Thrombo-Angiitis Obliterans. Clinical, Physiologic and Pathologic Studies. By GEORGE E. BROWN and EDGAR V. ALLEN, Division of Medicine, Mayo Clinic, Collaborating in Pathology with HOWARD R. MAHORNER, Fellow in Surgery, The Mayo Foundation. 219 pages, 61 figures, 12 tables, and 1 colored plate. W. B. Saunders Company, Philadelphia, 1928. Price in cloth: $3.00.

This study is based on observations made on more than 300 cases of thrombo-angiitis obliterans seen in the Mayo Clinic in the years 1922 to 1927 inclusive. Each year has seen an increase in the number of cases recorded in this clinic; at present the ratio of patients with this disease to all males registering is approximately 1:400. The study includes the pathologic examination of fifty amputated specimens, twenty-seven

of which were studied by Mahorner. The comparatively recent recognition of the disease, its peculiar sex and age frequency, together with the racial predilection and the probability of its increasing incidence have led to a greatly increased interest in this disease. There are thirteen chapters in the book, treating of the history of the disease, its etiology and pathology, clinical course and clinical types, analysis of symptoms, erroneous interpretations of symptoms, history taking and examination, diagnosis, miscellaneous studies, treatment, prognosis and special methods of investigation. These chapters are followed by a bibliography and index. In the first few years of the authors' experience practically all of the cases observed were associated with severe degrees of gangrene. Owing to a wider appreciation of the existence of thrombo-angiitis by the medical profession a larger number of cases are now seen in the stages before the appearance of trophic disturbances and gangrene. The institution of prophylactic and physical measures in the early stages of the disease, the realization of the importance of rest, the use of nonspecific protein therapy, and the application of surgical measures to the sympathetic nervous system in selected cases, have prevented many amputations and have afforded a more optimistic prognosis. The etiologic agent in thrombo-angiitis obliterans is still unknown; and more work remains to be done on the problems of anoxemia in relation to claudication and thrombosis. This volume is a valuable contribution to the subject of vascular diseases; and it cannot fail to be interesting and valuable to both internist and surgeon. The various chapters are thoroughly up to date and constitute a very complete description of the disease. Of interest is the well-known association of tobacco smoking, and the probabilities of the increase of the disease due to the present wide-spread abuse of this habit. The book is clearly and concisely written, the illustrations are to the point and are most useful. The authors believe that the disease primarily falls into the realm of the internist, but that the close association of surgeons and internists is important.

Nephritis. By HERMAN ELWYN, M.D., Assistant Visiting Physician, Gouverneur Hospital, New York. 347 pages, 2 figures. The MacMillan Company, New York, 1929. Price in cloth: $4.00.

This is a reprinting of this work on nephritis which was first publlished in 1926. In it Elwyn presents the individual forms of nephritis with an attempt at correlation of the clinical phenomena with the pathologic changes. As a necessary preliminary to such a presentation he discusses the normal function of the kidney, renal insufficiency, hypertension and uremia, which make up the first four chapters. Chapter five is concerned with the classification of nephritis; chapter six with embolic glomerulonephritis; chapter seven with non-embolic glomerulonephritis; chapter eight with acute diffuse glomerulonephritis; chapter nine treats of the subacute and subchronic forms of diffuse glomerulonephritis; and chapter ten with chronic diffuse glomerulonephritis. The remaining chapters discuss the kidney of pregnancy, lipoid nephrosis, amyloid nephrosis, the bichloride kidney, the pathology, etiology and pathogenesis of arteriosclerosis, renal arteriosclerosis with and without renal insufficiency and the syndrome of benign hypertension. In discussing the kidney of pregnancy the author ventures to offer a new explanation for the occurrence of eclampsia in pregnancy. Elwyn follows Volhard very closely in the correlation of the fundamental pathologic and chemical changes in the various forms of nephritis with the clinical phenomena. During the last twenty-five years our knowledge of nephritis has been much advanced; in this book this advance of knowledge on this important subject is brought together and very clearly presented. It is a thoroughly practical and valuable presentation of the subject from the modern standpoint.

Spinal Anesthesia. (Subarachnoid Radicular Conduction Block), *Principles and Technique.* By CHARLES H. EVANS, M.D.,

Clinical Assistant, N. Y. Postgraduate Medical School and Hospital, Lying-in Hospital of the City of New York; Assistant Surgeon, N. J. Orthopedic Hospital, Orange. Introduction by W. Wayne Babcock, M.D., F.A.C.S. Foreword by Charles Gordon Heyd, M.D., F.A.C.S. 203 pages, 41 illustration, 3 in color, one folding colored plate. Paul B. Hoeber, Inc., New York, 1929. Price in cloth: $5.50 net.

In this volume the author attempts a survey of the literature which has appeared upon this subject, and combines with it the results of his own personal experience in spinal anesthesia. In spite of the large amount of research and the numerous articles upon spinal anesthesia that have appeared during the last twenty-five years, there has been very little effort made to put the present knowledge of the subject into concise form available to the medical profession. There are excellent summaries on spinal anesthesia which are lost to the profession because the literature containing the material is found scattered throughout the specialties of medicine. The original purpose of the author was to collect data for a short manuscript to be used as a guide at the N. Y. Postgraduate Hospital. The patients undergoing surgical operations under this form of anesthesia were carefully studied, especially as to the psychologic changes taking place both at the time of operation and afterwards, the immediate physiochemical relations and the postoperative convalescence. Further study led to this monograph, which the author hopes will be the means of aiding other surgeons who may desire to employ spinal anesthesia in their practice. The author is convinced that not only is spinal anesthesia safe, but that it is the most satisfactory of all forms of anesthesia for certain surgical procedures. He has selected efficient technical methods from the many advocated, and this book will be found of great value by those who would prepare themselves for the use of spinal anesthesia. Its perusual will give all necessary information as to when, how and why the surgeon should employ spinal anesthesia.

Thyroxine. By Edward C. Kendall, M.S., Ph.D., D.Sc., The Mayo Foundation, Rochester, Minnesota. American Chemical Society Monographs. 225 pages. The Chemical Catalog. Company, Inc., New York, 1929. Price in cloth: $5.50.

This volume is an American Chemical Society Monograph which was advertised in 1919, but because of the prolongation of the chemical investigation of thyroxine appears now as the forty-seventh of the published series of monographs. Much of the work which is presented, however, has been completed in the last two or three years, and any monograph on the chemistry of the thyroid prepared before the present year would have been out of date almost before the printing had been completed. What has been assembled in the monograph has been chosen from the viewpoint of the chemist and with a desire to correlate the activity of the thyroid gland with reactions which involve oxidation. The story of thyroxine is told in this monograph up to the time of its production in a pure form by the English observers, Harington and Barger, in 1927, who showed that thyroxine is a tetra-iodo derivative of oxyphenyl tyrosine, and were able to produce it synthetically. These observers also showed that the physiological activity of the synthetic product is identical with that of the natural product. Thyroxine has a definite place in the study of normal and abnormal mental processes. Whether or not it functions as an enzyme or influences the velocity of enzyme may be a matter for controversy, but the processes which it does affect can be shown to be of fundamental importance, not only to the physiologic chemist but to the social and economic activities of man. The theoretical deductions in this monograph have been largely based on the goiter work of the Mayo Clinic. The author himself regards it "as the photograph of an ever-changing scene." There are many questions left unanswered; time will bring many additions and possibly many alterations.

College News Notes

PROGRAM
THIRTEENTH ANNUAL CLINICAL SESSION

BOSTON COMMITTEES

JAMES H. MEANS, *General Chairman*

COMMITTEE ON ARRANGEMENTS

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COMMITTEE ON HALL

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PRELIMINARY PROGRAM
ANNUAL CLINICAL SESSION
THE AMERICAN COLLEGE OF PHYSICIANS
APRIL 8-12, 1929

Monday, April 8, 1929

OPENING SESSION, 2:30 O'CLOCK

Hotel Statler Ballroom

1. Addresses of Welcome. David L. Edsall, Dean of Harvard Medical School. Alexander S. Begg, Dean of Boston University Medical School. A. Warren Stearns, Dean of Tufts College Medical School. John M. Birnie, President of Massachusetts Medical Society. Lincoln Davis, President of Suffolk District Medical Society.

2. Reply to Addresses of Welcome. Charles F. Martin, President of The American College of Physicians.

3. Tuberculosis: A Confession of Faith. Lawrason Brown, Saranac Lake, N. Y.

4. (Title not yet announced.) Lewellys F. Barker, Baltimore.

5. Juvenile Diabetes. I. M. Rabinowitch, Montreal.

6. Glycosuria. James E. Paullin, Atlanta.

7. Clinical Aspects of Paroxysmal Hypertension. M. C. Pincoffs, Baltimore.

EVENING SESSION, 8:00 O'CLOCK
Hotel Statler Ballroom

Symposium on Deficiency Diseases

1. The Fundamental Nature of Deficiencies. George R. Minot, Boston.

2. Pathology of Deficiencies. S. Burt Wolbach, Boston.

3. Biochemistry and Physiology of Deficiencies. George R. Cowgill, New Haven.

4. Pernicious Anemia. Randolph West, New York.

Tuesday, April 9, 1929
MORNING, 9:00 TO 12:00 O'CLOCK
Hospital Clinics

AFTERNOON, 2:30 TO 5:00 O'CLOCK
Hotel Statler Ballroom

1. Fatigue and Infection. W. L. Holman, Toronto.

2. Neoplasms. J. B. Murphy, New York.

3. Specific Dynamic Action of Protein, Fat and Carbohydrate in Altered States of Nutrition. Edward H. Mason, Montreal.

4. The Relation of Neisserian Infection to the Various Types of Arthritis. O. H. Perry Pepper, Philadelphia.

5. The Fallacy of Vaccine Therapy. Charles C. Bass, New Orleans.

6. The Treatment of Angina Pectoris. Harlow Brooks, New York.

7. The Coronary Problem. Arthur R. Elliott, Chicago.

8. Clinical Aspects of Trichiniasis. Lewis A. Conner, New York.

9. An Intensive Clinical Study of a Graphic Method of Recording Blood Pressure. Louis F. Bishop and Louis F. Bishop, Jr., New York.

EVENING SESSION, 8:00 O'CLOCK
Hotel Statler Ballroom

1. Psychiatry in Relation to Medicine. Austin F. Riggs, Stockbridge, Mass.

2. Syphilis of the Adrenals and Its Relationship to the So-called Idiopathic Addison's Disease. Aldred S. Warthin, Ann Arbor.

3. Lung Syphilis. R. I. Rizer, Minneapolis.

A *smoker* will follow this session.

Wednesday, April 10, 1929
MORNING, 9:00 TO 12:00 O'CLOCK
Hospital Clinics

AFTERNOON, 2:30 O'CLOCK
Hotel Statler Ballroom

1. The Treatment of General Paresis. Harry C. Solomon, Boston.

2. Psychiatry's Part in Preventive Medicine. Arthur H. Ruggles, Providence.

3. The Need of Emotional Data in the Medical History. John Favill, Chicago.

4. Milder Forms of Coronary Obstruction. James B. Herrick, Chicago.

5. The Failing Heart of Middle Life. David Riesman, Philadelphia.

6. Hypertension. George C. Hale, London, Ont.

7. Undulant Fever in the United States. George Blumer, New Haven.

8. (Title not yet announced.) Robert A. Cooke, New York.

9. Tobacco Smoking and Gastric Symptoms. Irving Gray, Brooklyn.

EVENING SESSION, 8:00 O'CLOCK
Hotel Statler Ballroom

1. Serums and Vaccines in the Prevention and Treatment of Disease. Benjamin White, Boston.

2. Clinico-Roentgenological Conference. M. C. Sosman and Associates, Boston.

Thursday, April 11, 1929
MORNING, 9:00 TO 12:00 O'CLOCK
Hospital Clinics

AFTERNOON, 2:30 O'CLOCK
Hotel Statler Ballroom

1. The Treatment of Acute Asphyxia. Cecil K. Drinker, Boston.

2. The Significance of Abnormal Metabolic Features in the Management of Thyrotoxicosis. Walter W. Palmer, New York.

3. Can or Will the Internist Practice Preventive Medicine? George H. Bigelow, Boston.

4. Factors in the Prognosis of High Blood Pressure. W. W. Herrick, New York.

5. The Carotid Sinus Reflex (Hering); Its Use in the Diagnosis and Treatment of Certain Cardiovascular Diseases. C. Saul Danzer, Brooklyn.

6. Lead Poisoning from Snuff. Raymond J. Reitzel, Galveston.

The General Business Meeting of The College will be held at 4:00 in the Hotel Statler Ballroom. All Masters and Fellows should attend.

EVENING, 7:00 O'CLOCK

Annual Banquet of The College
To be followed by a Dance.

Address: George E. Vincent, President of Rockefeller Foundation.

Friday, April 12, 1929
MORNING, 9:00 TO 12:00 O'CLOCK
Hospital Clinics

AFTERNOON, 2:30 O'CLOCK
Hotel Statler Ballroom

1. Motion Picture: Demonstrating Its Value in Teaching Electrocardiographic Interpretations of Cardiac Arrythmias. Joseph B. Wolffe, Philadelphia.

2. Dr. William Dunlop and Pioneer Canadian Medicine. J. W. Crane, London, Ont.

3. Rheumatic Fever. Homer F. Swift, New York.

4. (Title not yet announced.) J. C. Meakins, Montreal.

5. Results to Be Expected in Malignant Disease Treated by Radiotherapy. George E. Pfahler, Philadelphia.

6. The Problem of the Nervous Patient. Charles H. Nielson, St. Louis.

7. Endogenous Obesity—A Misconception. L. H. Newburgh and M. W. Johnston, Ann Arbor.

EVENING SESSION, 8:00 O'CLOCK
Hotel Statler Ballroom
Convocation Exercises

The General Profession is cordially invited. No special admission tickets are required.

1. Convocation Ceremony.

2. President's Address. Charles F. Martin, Montreal.

PRELIMINARY PROGRAM OF SPECIAL CLINICS AND DEMONSTRATIONS

This year the general session will be held in the afternoons and evenings, while clinics and demonstrations will be held in the mornings from 9:00 to 12:00.

Special Admission Cards required. Clinic reservation forms and full directions will accompany the Final Program. Reservations may be made by mail or daily at the Registration Bureau.

Special clinics and demonstrations will be held as follows:

A

BETH ISRAEL HOSPITAL

Program in charge of Herrman L. Blumgart

B

BOSTON CITY HOSPITAL

1. (A guest will give a clinic at this time; the name will be announced later.)

2. The Progress of the Boston City Hospital. John J. Dowling, Superintendent.

3. Treatment of Pneumonia. Demonstration of Cases. Edwin A. Locke.

4. Clinic of Unusual Cases. Francis W. Palfrey.

5. Pernicious Anemia. Demonstration of Cases. William B. Castle.

6. Treatment of Anemias. Demonstration of Cases. George R. Minot.

Wednesday, April 10, 1929

1. (A guest will give a clinic at this time; the name will be announced later.)

2. Gastro-Intestinal Cases. Franklin W. White.

3. Cardiac Cases. William H. Robey.

4. Nephritis Cases. William R. Ohler.

5. The Surgical Treatment of Pulmonary Tuberculosis. Demonstration of Cases. Edward D. Churchill.

Hypertension and Arteriosclerosis. Demonstration of Cases. Soma Weiss.

Thursday, April 11, 1929

1. Cardiac Cases. Edward N. Libby and Thomas J. O'Brien.

2. A Case Illustrating the Value of the Electrocardiogram. James M. Faulkner.

3. Epilepsy. William G. Lennox.

4. Diseases of the Coronary Vessels. Demonstration of Cases. Joseph T. Wearn.

5. Peptic Ulcer. Demonstration of Cases. Maurice Fremont-Smith.

6. Neurological Cases. Stanley Cobb.

7. (A guest will give a clinic at this time; the name will be announced later.)

Friday, April 12, 1929

1. (A guest will give a clinic at this time; the name will be announced later.)

2. Cases of Disease of the Hemopoietic System. Ralph C. Larrabee.

3. Lymphoblastoma. Demonstration of Cases. Henry Jackson, Jr.

4. Tropical Diseases. Demonstration of Cases. George C. Shattuck.

5. Fluoroscopic Diagnosis in Chest Conditions. Demonstration of Cases. Harold W. Dana.

6. Carcinoma of the Head of the Pancreas. Demonstration of Cases. Irving J. Walker.

C

BOSTON CITY HOSPITAL
THORNDIKE MEMORIAL LABORATORY

Wednesday and Thursday
April 10 and 11
Between 10:30 and 12:30

Demonstration of Researches Concerning the Following Topics

Dr. Castle and Associates	Anemia
Dr. Jackson and Associates	Malignant Tumors
Dr. Lawrence and Associates	The Physiology and Pathology of White Cells
Dr. Lennox	Epilepsy
Dr. Minot and Associates	The Blood
Dr. Nye and Associates	Bacteriological Problems
Dr. Wearn and Associates	The Capillaries
Dr. Weiss and Associates	Vascular Problems

BOSTON CITY HOSPITAL
SOUTH DEPARTMENT

Program in charge of Edwin H. Place

Ward visits on (1) diphtheria, (2) scarlet fever, (3) a few of the other minor groups such as chicken pox, mumps, measles and whooping cough.

Amphitheater demonstration of cases of chronic laryngeal injury and other damages resulting from contagious diseases.

E

BOSTON DISPENSARY

TUESDAY, APRIL 9, 1929

1. Heart Disease. David Davis.
2. Essential Hypertonia. David Ayman.
3. Neurological Clinic. A. Warren Stearns.
4. Obesity. Mark Falcon-Lesses.
5. Gastro-Intestinal Clinic. Percy B. Davidson.

WEDNESDAY, APRIL 10, 1929

1. Bronchiectasis. William Dameshek.
2. Psychalgia. Joseph H. Pratt.
3. Arthritis. John D. Adams.
4. Chronic Pancreatic Disease. Bert B. Hershenson.
5. Tuberculosis. H. Louis Kramer.

THURSDAY, APRIL 11, 1929

1. Neurosyphilis. Arthur Beck.
2. Neurasthenia. Joseph H. Kaplan.
3. Nephrosis. Tobert W. Buck.
4. Domiciliary Medicine in Clinical Teaching—Selected Case. Osadore Olef.
5. Domiciliary Medicine in Clinical Teaching—Selected Case. Charles Korb.
6. Diabetes. James H. Townsend.

F

CHILDREN'S HOSPITAL

Program in charge of Kenneth D. Blackfan

G

HOMEOPATHIC HOSPITAL
EVANS MEMORIAL CLINIC

TUESDAY, APRIL 9, 1929

1. Sterility Clinic. Special Emphasis to be Placed on the Constitutional Factors in Sterility. S. R. Meaker and A. W. Rowe.

WEDNESDAY, APRIL 10, 1929

Endocrine Clinic

1. Endocrine Diagnosis and Therapy. Charles H. Lawrence.
2. Endocrine Disorders Associated with Otosclerosis and the Menière Syndrome. D. W. Drury.
3. Eye Findings in Endocrine Disorders. W. D. Rowland.
4. Cases Presenting Outward Evidence of Endocrine Disorders Found on Study not to have Endocrine Disturbance. A. W. Rowe.
5. Dementia Praecox. L. G. Hoskins.
6. The Follicular Hormone. J. C. Janney.
7. Discussion on Sugar Metabolism as Influenced by Insulin in Pituitary Disease. H. Ulrich and A. W. Rowe.

THURSDAY, APRIL 11, 1929

General Medical Clinic

1. Heart Clinic. W. D. Reid.
2. Intestinal Migraine. C. W. McClure.
3. Neurology. N. H. Garrick.
4. Lung Abscess, Diagnosis and Treatment: Bronchoscopy, the Use of the Bronchoscope in Diagnosis and Treatment. L. R. Johnson.

FRIDAY, APRIL 12, 1929

(Program to be announced later.)

H MASSACHUSETTS GENERAL HOSPITAL

1. Clinic by James E. Paullin, Atlanta.
2. Thoracic Clinic. Frederick T. Lord.
3. Cases of Hypertension. William B. Breed.
4. Cardiac Clinic. Howard B. Sprague.
5. Endocrine Clinic. Walter Bauer and Dwight L. Sisco.

WEDNESDAY, APRIL 10, 1929

1. Clinic by Lewellys F. Barker, Baltimore.
2. Demonstration of Medical Cases. William B. Robbins.
3. Pediatric Clinic. Fritz B. Talbot and Harold L. Higgins.
4. Clinico-pathological conference. Richard C. Cabot and Tracy B. Mallory.
5. Diabetic Clinic. Roy R. Wheeler.

THURSDAY, APRIL 11, 1929

1. Clinic by O. H. Perry Pepper, Philadelphia.
2. Neurological Clinic. James B. Ayer.
3. Psychotherapy of Gastro-Intestinal Diseases. William Herman.
4. Gastro-Intestinal Clinic. Chester M. Jones.
5. Indications for Splenectomy. Arlie V. Bock.
6. Cases of Pernicious Anemia. Wyman Richardson.

FRIDAY, APRIL 12, 1929

1. Clinic by J. C. Meakins, Montreal.
2. Demonstration of Cases. Gerald Blake.
3. Medical Clinic. James H. Means.
4. Demonstration of Cases. F. Dennette Adams.
5. Anaphylaxis Clinic. Francis M. Rackemann.

I NEW ENGLAND BAPTIST HOSPITAL

Program in charge of Albert A. Hornor

J NEW ENGLAND DEACONESS HOSPITAL

Program in charge of Elliott P. Joslin

1. Carcinoma of the Colon and Colitis from the Surgical Point of View. Daniel F. Jones.
2. Gastro-Intestinal Cases. Sara M. Jordan and Chester Kiefer.
3. Thyroid Cases. Frank H. Lahey.
4. Pedigreed Diabetics. Elliott P. Joslin.
5. Surgery in Diabetics. L. S. McKittrick.
6. The Pathology of Diabetes. Shields Warren.

There will be further additions to this program including clinics by larynologists, ophthalmologists, gynecologists and roentgenologists.

K PETER BENT BRIGHAM HOSPITAL

1. Diagnosis of Certain Forms of Heart Disease. Lewis A. Conner, New York.
2. Chronic Myocardial Disease. Henry A. Christian.
3. Results of Treatment of Duodenal Ulcer. E. S. Emery.
4. Some Considerations on the Relation of Cardio-Renal System to Surgery of the Urinary Organs. William S. Quinby.
5. Bronchoscopy in Lung Disease. Lyman C. Richards.

WEDNESDAY, APRIL 10, 1929

1. Cardiac Disease, the Result of Infectious Processes. James B. Herrick, Chicago.
2. Gallbladder Disease. Channing Frothingham.
3. Bronchial Asthma. I. Chandler Walker.
4. Anemia. William P. Murphy.
5. Thrombophlebitis. John Homans.

THURSDAY, APRIL 11, 1929

1. Mitral Stenosis. David Riesman, Philadelphia.
2. Signs of Persisting Infection in Acute Rheumatic Fever. Clifford L. Derick.
3. Hemorrhagic Nephritis. James P. O'Hare.
4. A Surgeon's Views of the Treatment of Peptic Ulcer. David Cheever.
5. Neurosurgical Conditions. Harvey Cushing.

FRIDAY, APRIL 12, 1929

1. Hypertension. Charles F. Martin, Montreal.
2. Vascular Disease in Diabetes Mellitus. Reginald Fitz.
3. Treatment of Certain Types of Cardiac Arrhythmia. Samuel A. Levine.
4. Treatment of Trifacial Neuralgia. Gilbert Horrax.
5. Diuretics. Henry A. Christian.

L

ROBERT BRECK BRIGHAM HOSPITAL

Program in charge of Louis M. Spears
Clinics on Arthritis

M

UNITED STATES NAVAL HOSPITAL

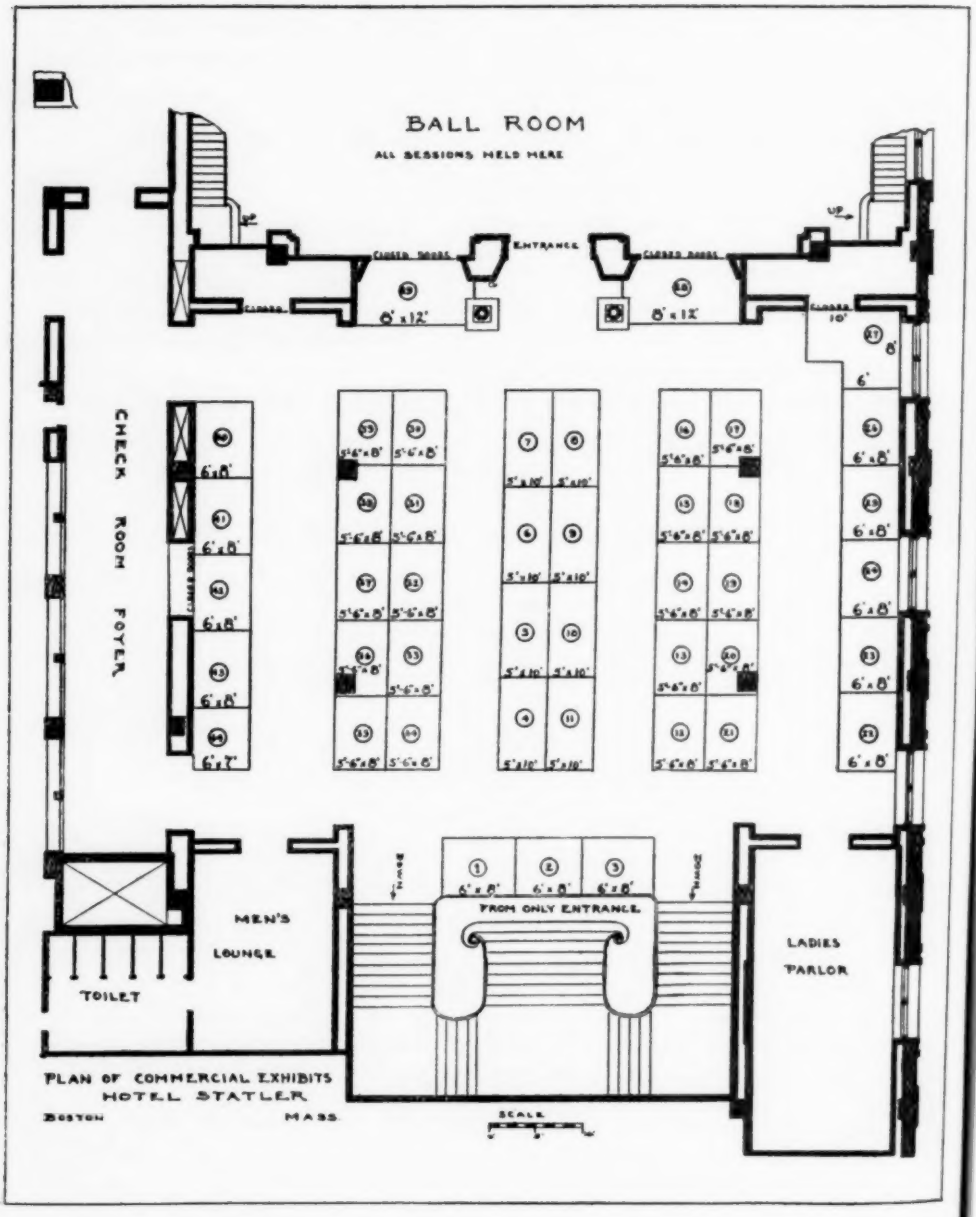
Program in charge of Capt. F. L. Pleadwell, M.C., U. S. N.

Presentation of medical cases in the conference room of the hospital each morning. Following this the group will be split up in sections of five. Each section will be in charge of a ward medical officer, and the balance of the morning will be devoted to ward rounds.

TECHNICAL EXHIBIT

The technical exhibits have been arranged by the Executive Secretary, Mr. E. R. Loveland, and the following chart shows the arrangement of booths and the assignment to exhibitors from various parts of the country. The exhibits are highly diversified in their variety and will bring to the attendants at the Clinical Session, the latest and most improved equipment, the best pharmaceutical products, almost the whole library of medical publications and many other products of special interest to the Internist, Pediatrician, Neurologist, Psychiatrist, Radiologist and research worker.

This Exhibit is undoubtedly the best arranged and the most popular one that The College has yet had. The location is in the Ballroom Foyer where all attendants to the meeting will pass through the exhibits daily. The Joseph T. Griffin Decorating Company, of Louisville, Kentucky, who installed the exhibits for the American Medical Association, the Southern Medical Association and many other prominent medical societies, will be in charge of the booths and decorations.


BALL ROOM
ALL SESSIONS HELD HERE
ENTRANCE
CLOSED DOORS
CLOSED
UP
8' x 12'
8' x 12'
10'
6'
CHECK ROOM FOYER
MEN'S LOUNGE
TOILET
FROM ONLY ENTRANCE
LADIES PARLOR
PLAN OF COMMERCIAL EXHIBITS
HOTEL STATLER
BOSTON MASS.
SCALE

LIST OF EXHIBITORS

Space	Name	City and State	Product
20	Abbott Laboratories	North Chicago, Ill.	Pharmaceutical Products
12 & 21	D. Appleton & Company	New York, N. Y.	Medical Publications
31	The Battle Creek Food Company	Battle Creek, Mich.	Health Foods
22	Bausch & Lomb Optical Co.	Rochester, N. Y.	Microscopes, Photomicro & Projection Apparatus
40	P. Blakiston's Son & Co.	Philadelphia, Pa.	Medical Publications
13	The Borden Sales Company, Inc.	New York, N. Y.	Merrell Soule Infant Foods
26	Britesun, Inc.	Chicago, Ill.	Therapeutic Lamps
25	Cambridge Instrument Co., Inc.	New York, N. Y.	Electrocardiographs & Accessories, and other Physiological Instruments
3	Cameron's Surgical Specialty Co.	Chicago, Ill.	Electro-Diagnostic Surgical & Dental Instruments
44	G. W. Carnrick Co.	Newark, N. J.	Pharmaceutical Products
1	Warren E. Collins, Inc.	Boston, Mass.	Metabolism and Oxygen
49	Davies, Rose & Co., Ltd.	Boston, Mass.	Trethylene, Pil. Digitalis, Shadocal
14	F. A. Davis Company	Philadelphia, Pa.	Medical Publications
16	Deshell Laboratories, Inc.	Chicago, Ill.	"Petrolagar"
42 & 43	General X-Ray Company	Boston, Mass.	"Morse" Wave Generator, GX-Galvine-Faradie Plate, Diathermy Apparatus, Electrodes
34	Paul B. Hoeber, Inc.	New York, N. Y.	Medical Publications
19	Horlick's Malted Milk Corporation	Racine, Wis.	Malted Milk Products
17	Kalak Water Company, Inc.	New York, N. Y.	Kalak Water
4	Charles B. Knox Gelatine Co., Inc.	Johnstown, N. Y.	Knox Gelatine
15	Lavoris Chemical Company	Minneapolis, Minn.	"Lavoris"
45	LaMotte Chemical Products Co.	Baltimore, Md.	LaMotte Blood Chemistry Outfits
30	Lea & Febiger	Philadelphia, Pa.	Medical Publications
47	Lederle Antitoxin Laboratories	New York, N. Y.	Biological Products and Pharmaceutical Specialties
9	J. B. Lippincott Company	Philadelphia, Pa.	Medical Publications

29	MacGregor Instrument Company	Needham, Mass.	Vim Stainless Steel Needles; Vim Emerald Luer Syringes; Vim Surgical & Medical Specialties
8	The Macmillan Company	New York, N. Y.	Medical Publications
2	E. F. Mahady Company	Boston, Mass.	Anaesthetic Apparatus, Laboratory Equipment, Diagnostic & Scientific Apparatus, Vaccines, Intravenous Products, Orthopedic Appliances & Supplies, Instruments for Operating Room, E.F.M. Catgut
18	The Medical Protective Company	Chicago, Ill.	Malpractice Insurance
24	Mellin's Food Company	Boston, Mass.	Mellin's Food
38	Merck & Company, Inc.	Rahway, N. J.	Pharmaceutical Products
23	The Wm. S. Merrell Company	Cincinnati, Ohio	Pharmaceutical Products
32 & 37	Merrell Soule Company	New York, N. Y.	Infant Foods
35	The C. V. Mosby Company	St. Louis, Mo.	Medical Publications
39	Thomas Nelson & Sons	New York, N. Y.	Medical Publications
28	The E. L. Patch Company	Boston, Mass.	Cod Liver Oil
6	Pittsburgh Plate Glass Co.	Pittsburgh, Pa.	"Helioglass"
7	Richards, Inc.	Glenolden, Pa.	Psyllium Seed & Acidophilus Products
11	W. B. Saunders Company	Philadelphia, Pa.	Medical Publications
27	Sanborn Company	Cambridge, Mass.	"Graphic" Metabolism Apparatus
41	Spencer Lens Company	Boston, Mass.	Optical Instruments, Projection Apparatus, Laboratory Equipment
50	Swan-Myers Co.	Indianapolis, Ind.	Pollens, Ephedrine Preparations, Dextrose Ampoules and other Pharmaceutical Products
36	Tailby-Nason Company	Boston, Mass.	Cod Liver Oil
51	Taylor Instrument Companies	Rochester, N. Y.	"Tycos" Sphygmomanometer, Thermometers and Hygrometers
5 & 10	Victor X-Ray Corporation	Chicago, Ill.	Electrocardiograph & Quartz Lamps
46	Vitaglass Corporation	New York, N. Y.	Vita Glass
33	Winthrop Chemical Company, Inc. H. A. Metz Laboratories, Inc.	New York, N. Y.	Pharmaceutical Products

BAUSCH & LOMB CO.

Booth 22

In each issue of this publication for the past few months, this space has been devoted to a brief description of a partial list of the optical instruments which Bausch & Lomb manufacture for the medical Profession.

At the American College of Physicians meeting, April 8-12, 1929, the instruments which have been described herein will be on exhibit. A list of the instruments to be exhibited are:

Microscopes	Hemoglobinometers
Microtomes	Centrifuges
Colorimeters	Haemacytometers

The physician who is contemplating buying new instruments will find this exhibition an excellent place to make his choice. Undoubtedly some who do not intend to purchase will be able to obtain a great deal of useful information on new instruments and improved methods, which should aid them in their practice.

Literature which will give a comprehensive knowledge of the latest developments in the field can be had at the Bausch & Lomb booth. If, however, you do not attend the meeting, the literature will be sent to you upon application to the Company.

P. BLAKISTON'S SON & CO.

Booth 40

The translation of Kaufmann's Pathology by Dr. Stanley P. Reimann, published by Blakiston in January, makes available to all American physicians and scientists a work of international repute, whose usefulness has been confined previously to those having a high aptitude for scientific German. The work is a most complete human pathology, general and special. Subjects of most importance to the practicing physician are emphasized and given in great detail. The work is in three volumes and contains 1072 illustrations, those of the last German edition being amplified by many unique pictures drawn by the staff artist at the Lankenau Hospital. A descriptive prospectus will be sent by the publisher upon request.

THE BORDEN SALES COMPANY, Inc.

Booth 13

In 1856 the first successful process for condensing milk was patented by Gail Borden. Today, 73 years later, the annual world production of all forms of concentrated milk amounts to more than three billion pounds, of which approximately 60 per cent is manufactured in the United States. The Borden Company continues to be the leading producer and distributor of condensed, evaporated, powdered and malted milks, and other milk products.

DESHELL LABORATORIES, Inc.

Booth 16

Prominent among the exhibits at the next Clinical Session will be Petrolagar—the emulsion of mineral oil.

Samples and literature pertaining to the wide application and usefulness of Petrolagar may be obtained by addressing 536 Lake Shore Drive, Chicago, Illinois.

THE WM. S. MERRELL COMPANY

BOOTH 23

This is the new symbol of the Wm. S. Merrell Company as they have begun their second century in the preparation of medicines for the use of the medical profession. This symbol is the sign used by the ancient alchemists to represent that by which perfection may be attained, the accomplishment of the ideal, the reward to those who faithfully follow the laws of their Philosophy.

The Merrell Symbol was also used by the Alchemists to represent the Elixir of Life, which, if taken according to instructions, would heal the sick and renew the life of the old. Needless to say, such an Elixir remained as an ideal and was never found. The development of rational Therapeutic agents in the closest approximation of the ideal that has as yet been made.

SANBORN COMPANY

— 27 —

METABOLISM testing with simplified apparatus giving reliable data for diagnosis will be demonstrated at Space 27 by Sanborn Company. This Company manufactures and supplies directly to physicians and hospitals. Medical conventions afford special opportunity for Sanborn owners and prospective owners to get practical information of the latest and best methods of testing. The new and approved Sanborn ELECTROCARIOGRAF—portable and transportable models—the least expensive but the best, will gladly be explained. Visit Space 27.

TAYLOR INSTRUMENT COMPANY

— 51 —

One of the exhibits by the Taylor Instrument Companies will be the Tycos Sphygmomanometer.

The Tycos Recording Sphygmomanometer introduces a means of measuring blood pressure which necessitates neither stethoscope nor indicator, its automatic nature insuring an exactness of systolic and diastolic pressure points never before attained.

In addition, any cardiac irregularity producing characteristic changes in the brachial artery is identified by the Tycos tracing.

This is best realized by an inspection of the actual graphs of various pathologies, which are included in their latest booklet, "The Clinical Use of the Tycos Recording Sphygmomanometer." Copy will be sent upon request.

SUBSCRIPTION DINNER AND MEETING of the SOUTHERN CALIFORNIA MEMBERS of the AMERICAN COLLEGE OF PHYSICIANS

The Southern California Fellows and Associates of the American College of Physicians gathered together at dinner on Friday evening, January 18, at the California Club, Los Angeles. Among those present were Dr. Leonard Rowntree, Rochester, Minnesota; Dr. William Engelbach, formerly of St. Louis, Mo.; Doctors Samuel Alter, Samuel Ayres, Charles C. Browning, Ralph L. Byrnes, John V. Barrow, William E. Costolow, Egerton Crispin, Roland Cummings, Leland S. Chapman, Harry W. Coffin, Robert Cunningham, Kenneth Davis, Ernest Fishbaugh, Kendal Frost, George G. Hunter, Herold P. Hare, Carl R. Howson, Samuel I. Ingham, Joseph M. King, Henry H. Lissner, Ross Moore, Thomas J. Orbison, Francis M. Pottenger, George Piness, Leon Shulman, John M. Shuman, Harold H. Smith, Frederick Speik, Earl Sweet, Roy E. Thomas, Raymond G. Taylor, Giuseppe Vercellini, Walter Wessels, Edward R. Ware, and John C. Webster, Los

Angeles; Frederick B. Clarke, Long Beach; Edward W. Hayes, Monrovia; Walter P. Bliss, Joseph D. Condit, John S. Hibben, Arthur T. Newcomb, Robert E. Ramsay, Stephen Smith, Willard J. Stone and John M. Wilson, Pasadena; Paul E. Simonds, Riverside; George S. Langdon, San Bernadino; Hilmar O. Koefod, Santa Barbara; Lyle C. Kinney, Chancel L. Lounsberry and Robert Pollock, San Diego.

Following dinner Dr. Egerton Crispin of the Board of Governors spoke on "Purposes and Ideals of the College." Dr. F. M. Pottenger of the Board of Regents spoke on "The Future Plans of the College." The medical address of the evening on "Recent Advances in our Knowledge of the Liver" was given by Dr. Leonard Rowntree, Director of Medical Service, Mayo Clinic, and Professor of Medicine in the University of Minnesota.

Dr. Robert Bernhard Becomes Life Member

Announcement is made that Dr. Robert Bernhard (Fellow), New Orleans, Louisiana, has become a Life Member of the American College of Physicians by subscribing to the Life Membership Endowment Fund.

Dr. Noxon Toomey (Life Fellow), St. Louis, read a paper on "Pruritus and Dermatitis of Internal Origin" before the Lee County Medical Society, Fort Madison, Iowa, on December 20.

Dr. L. B. McBrayer (Fellow) is the newly elected President of the Southern Pines (N. C.) Chamber of Commerce.

Dr. William Fitch Cheney (Fellow), San Francisco, has been elected President of the Commonwealth Club of California for 1929. This is a large social service club with nearly 5000 members; and this is the first time in the twenty-five years of its existence that a physician has been elected its President.

Surgeon General Merritte W. Ireland (Fellow) of the U. S. Army, was the principal speaker at a meeting of the Indianapolis Medical Society, January 19, when they had as their guest Private John R. Gissinger, of Huntington, Indiana, who volunteered to be bitten by infected mosquitoes during the Yellow Fever experiments in Cuba following the Spanish American War.

Surgeon General Hugh S. Cumming (Fellow) of the U. S. Public Health Service spoke before the section on Public Health and Industrial Medicine of the Philadelphia College of Physicians on January 18.

Dr. Samuel Weiss (Fellow), New York, demonstrated the new so-called gastric camera before the American-Hungarian Medical Association at the New York Academy on Medicine on December 18. Dr. Weiss also demonstrated the gastric camera before one hundred physicians of New York City on December 22, the demonstrations being made on an inmate at the Sing Sing Prison.

Dr. Noxon Toomey (Life Fellow), St. Louis, was recently elected Twelfth Supreme Eminent Master of the Chi Zeta Chi Medical Fraternity at the Twenty-fifth Anniversary Convention held in St. Louis at the end of December.

Dr. Thomas G. Simonton (Associate), Pittsburgh, President of the Pennsylvania State Medical Society, Dr. Orlando H. Petty (Fellow), Philadelphia, Dr. H. R. M. Landis (Fellow), Philadelphia, and Dr. Elmer H. Funk (Fellow), Philadelphia, were speakers on the program at the annual meeting of the Pennsylvania Tuberculosis Society at Pottsville, January 15 and 16.

Dr. Kenneth M. Lynch (Fellow), Charleston, addressed the annual meeting of the Marlboro County Medical Society at Bennettsville (S. C.), January 10, on "Carcinoma of the Cervix Uteri."

Dr. Joseph C. Doane (Fellow), Philadelphia, addressed the Pennsylvania Conference on Social Welfare at Harrisburg during its Twenty-fifth Annual Session, February 13-16.

Dr. Solomon Solis Cohen (Fellow), Philadelphia, was the recipient of a gold medal at a dinner by the Phi Lambda Kappa Medical Fraternity, December 30, 1928.

Dr. Warren H. Newcomb (Associate) recently resigned as health officer of Morgan County, Illinois.

Dr. Walter M. Simpson (Fellow), Dayton, Ohio, addressed the Michigan Public Health Association at Lansing, January 9-10, on "Tularemia."

Dr. Arthur C. Morgan (Fellow), Philadelphia, was recently appointed Chairman of the disabled soldiers and medical aid committee of the Philadelphia County Council of the American Legion.

Surgeon General Merritte W. Ireland (Fellow) directed the recently completed history of the Army Medical Department during the World War. Colonel Frank W. Weed was Editor-in-Chief.

Dr. Noxon Toomey (Life Fellow), St. Louis, published in the November, 1928, number of the *British Journal of Dermatology and Syphilis* an introductory article on his treatment of psoriasis by means of colloidal gold preparations.

Dr. Orlando H. Petty (Fellow), Philadelphia, was installed as President of the Philadelphia County Medical Society on January 9.

Dr. Lewellys Barker (Fellow), Baltimore, was elected a Vice President for 1929 of the Pan-American Medical Association at its annual conference in Havana.

Dr. Leonard G. Rowntree (Fellow), Rochester, Minnesota, and Dr. Ernest C. Fishbaugh (Fellow), Los Angeles, addressed the Los Angeles County Medical Association January 17.

Dr. Lyell C. Kinney (Fellow), San Diego, addressed the San Bernardino County Medical Society recently.

Dr. George G. Richards (Fellow), Salt Lake City, addressed the San Diego County Medical Society recently on ulcer of the stomach.

Dr. Gerald B. Webb (Fellow), Colorado Springs, Colorado, delivered a lecture at the University of Maryland at Baltimore on January 3.

Among Fellows of the College who addressed the forty-fifth annual meeting of the Tri-States Medical Association (including Arkansas, Mississippi and Tennessee) at Memphis, February 6-8, were Dr. John H. Musser, New Orleans; Dr. Ray M. Balyeat, Oklahoma City; Dr. Frank Smithies, Chicago; Dr. Lawrason Brown, Saranac Lake; Dr. Arthur C. Christie, Washington; Dr. Henry K. Dunham, Cincinnati; and Dr. Leonard G. Rowntree, Rochester, Minnesota.

The American Public Health Association will hold its Fifty-Eighth Annual Meeting in Minneapolis, Minnesota, September 30 to October 5, 1929.

Dr. C. R. Jones (Fellow), Pittsburgh, delivered an address on "The Romance of Medicine" (illustrated), January 10, as one of a series of lectures sponsored by the Academy of Science and Art of Pittsburgh. Dr. Jones is Professor of Principles of Medicine at the University of Pittsburgh, and for many years has been Treasurer of the American College of Physicians.

Dr. E. A. Hines (Fellow), Seneca, S. C., was elected President of the South Carolina Pediatric Society at its annual meeting in Columbia, January 15. Dr. Hines is the Secretary Editor of the South Carolina Medical Association.

OBITUARY

Joseph Sailer, Fellow of the American College of Physicians, died at his home in Philadelphia as the last hours of 1928 were spending themselves. He had attended the New Orleans meeting of the College where he read a paper on "Conditions Contra-indicating the Use of Digitalis." Very few appreciated at the time what an effort it must have been to prepare for and make this presentation. Although it was a tedious journey for him, he returned home considerably refreshed and apparently much better for having experienced a week of Southern hospitality. He undertook, against advice, the trip to Minnesota last June where he presided over the sessions of the Section on Gastroenterology of the American Medical Association. This effort too must have distressed him more than his friends realized at the time. After his summer holiday, he resumed with difficulty his usual active life and continued to do so up until about two months before his death. Thus it was, as no doubt he wished it to be, that he continued his life busied almost to the end in loyal service to his profession and patients.

"Joe" Sailer as he was familiarly called by his friends and students was born in the City of Brotherly Love somewhat over 61 years ago of a father and grandfather who were prominent in the financial life of the city. It was hoped that he too would follow in the footsteps of his fathers. After graduating from the Biological Department of the University of Pennsylvania in 1886 and giving banking a trial of eight months, he decided however to study medicine, receiving his Medical Degree from the same University in 1891. Subsequent to this he spent four years as a resident in the Presbyterian and Philadelphia Hospitals in Philadelphia and as a student in Paris, Zurich and Vienna. During his European stay he worked in the laboratories of Déjerine, V. Monokow and Neisser. He was impressed at the time with the French school and especially enjoyed Charcot's instructions. Previous to entering the Medical School of the University of Pennsylvania he had spent a year in Germany learning her language.

After returning from Europe in 1895 he began to practice medicine as an associate of Dr. John Musser and at the same time became an Associate in Clinical Medicine in the Pepper Clinical Laboratory. He was at one time Assistant Pathologist at the Philadelphia Hospital and at other times Pathologist to the Pennsylvania Training School for Feeble Minded Children, Saint Joseph's and Maternity Hospitals in Philadelphia. He also taught in the Pathological Department of the University when Dr. Simon Flexner served as Professorship in this institution. In the Department of Medicine of his Alma Mater he rose from the position of Instructorship to that of a Clinical Professorship. At various times he was a physician to the Charity, Howard, Philadelphia, Policlinic, University and Presbyterian Hospitals.

He was interested in the entire field of Internal Medicine, but as his

friends know, he passed through three distinct periods in his medical life when his efforts were more or less concentrated on some special phase of the larger field. During the first of these periods he showed a neurological bent, during the second, gastro-enterology seemed to be uppermost in his mind, while the final years of his life were distinctly cardiovascular in their coloring. In this latter period he was especially concerned in the organization of the Philadelphia Association for the Study and Prevention of Heart Disease as well as the larger national body having the same objective in view. He was instrumental in bringing about the construction of a new home for convalescent cardiac children which opened its doors during his last illness. This event must have given him much satisfaction and the home serves as a splendid memorial to his interest in cardiac disease and more especially those who fall victims of this malady.

His bibliography in the main shows very clearly the three interests he had in internal medicine. In 1891 his essay on "Ouabain" won the Medical News Prize. When he returned from Europe he began to write articles chiefly concerned with neurological observations both of a clinical and pathological nature. "Alterations in the Spinal Cord in Azoturia," "A Contribution to the Pathology of the Choroid Plexus," "Degenerative Changes in Nerve Cells" and Meralgia Paresthetica" are some the contributions made during this first period. In 1904 his writings began to show his especial interest in the Gastrointestinal tract when the literature received his varied observations on gastric secretion, pancreatitis, carcinoma, achylia gastrica linitis plastica, visceroptosis and other subjects. From 1915 to the end, his major writings concerned the cardiovascular system. Scattered, however, throughout his writings appear papers concerning the field which was finally nearest his heart. Among these are articles dealing with aortic regurgitation, relative pulmonary insufficiency, aneurisms, the arrythmias and other titles concerned with this system.

During the world war he served with distinction and kept busily observing and giving to the medical profession the result of his studies. In this period appeared with others, a paper on pneumococcus carriers, one on mumps, one on cerebrospinal meningitis and another on the group consultant. His bibliography numbers at least 125 articles, among which are found a very interesting contribution "On the State of Medicine as Depicted by Pepys in the Reign of Charles II" and a thoroughly fine one on "Genghis Khan."

Besides his tremendous interest in medicine Dr. Sailer took much pleasure in the collection of books and line engravings. Of the latter he had accumulated a very noteworthy number of fine examples.

All who knew him were impressed by his fineness of character, by his gentility and by his tremendous fund of information which seemed to embrace every field of knowledge. With it all he was modest and never ostentatious. His undergraduate students loved him and respected him. This love and respect was held by his medical confreres whether they met him

later as postgraduate students, consultees, friends, or as fellow officers in the military service. His patients were loyal and devoted to him. They invariably appreciated his painstaking method and unusual skill as a doctor. He brought a tremendous vitality to his work. Even when very tired and in the last months when very sick he carried on with the spirit of one who found life exciting, important and full of humane interest. With all of his learning he was distinctly a man of action who found in the practice of his profession an outlet for an amazing energy that overflowed into other fields. American medicine has lost one of its most distinguished members and the American College of Physicians a great Fellow.

Dr. Truman G. Schnabel,
1704 Pine St.,
Philadelphia, Pa.

Dr. Frank William Fleischaker (Fellow), Louisville, Ky., died November 26, 1928, at St. Joseph's Infirmary, of heart disease following a recent surgical operation.

Dr. Fleischaker was born at Louisville, September 14, 1877. After graduation from the Louisville High School, he attended the Louisville College of Pharmacy, graduating in 1897; he then entered the University of Louisville, Medical Department, and received his medical degree in 1900. Immediately thereafter he went abroad for postgraduate medical study at the Universities of Heidelberg, Berlin and Vienna. He was professor of medicine at his alma mater from 1916 to 1925, when he became professor of clinical medicine at the same institution. This appointment he still held at the time of his death.

Dr. Fleischaker was a member of the Phi Chi medical fraternity, a member of the Jefferson County Medical Society, a member of the Kentucky State Medical Association and a Fellow of the American Medical Association. He was elected a Fellow of the American College of Physicians in 1920.

Annals of Clinical Medicine

There is a limited stock of complete volumes and odd lots of the 1922 to 1927 volumes of *Annals of Clinical Medicine* available for sale through the Executive Secretary's office at Philadelphia. Single copies of any number, where available in broken lots, will be sent postpaid for $.50.

It is also possible to secure any particular article that appeared in these five volumes in quantity by placing an order for reprinting through the Executive Secretary's office.

Year Book Omission

The following physician was omitted in error from the list of Associates, both alphabetical and geographical:

Elkourie, Haickel A. (I), 1230-35 Martin Building, Birmingham, Alabama; born '80; M.D. University of Nashville, Medical Department, 1901; Member, Jefferson County Medical Society, Alabama State Medical Society, Southern Medical Association, American Medical Association, and an Associate of the American College of Physicians; Licensed, Alabama, Kentucky, Tennessee.

Thirteenth Annual Clinical Session

of the

AMERICAN COLLEGE OF PHYSICIANS

BOSTON, MASS., APRIL 8–12, 1929

The 1929 Clinical Session will constitute one of the most important post-graduate weeks in the history of the College. The Harvard Medical School, the Boston University School of Medicine, Tufts Medical School, Boston City Hospital, Boston Dispensary, Children's Hospital, Homeopathic Hospital, Massachusetts General Hospital, Peter Bent Brigham Hospital, Robert Breck Brigham Hospital, Beth Israel Hospital, New England Baptist Hospital, New England Deaconess Hospital, U. S. Naval Hospital, Carney Hospital and the House of the Good Samaritan are cooperating and arranging programs. Eminent authorities from all parts of the country will participate in clinics, laboratory demonstrations, symposia and formal addresses. *See the College News Notes section for additional details of the program.*

Transportation: Over all railroads of the United States and eastern Canada on the Certificate Plan of reduced fares. Special trains from and to Chicago over the Michigan Central Railroad; special trains from and to Washington over the Pennsylvania Railroad and associated roads. Time-tables for special trains may be secured from ticket offices or passenger agents of roads mentioned, or from the Executive Secretary.

College Smoker, Tuesday evening, April 9; *College Banquet*, Thursday evening, April 11; *Convocation*, for the conferring of Fellowships, Friday evening, April 12.

Headquarters: Hotel Statler. Make reservations immediately.

For Information, address the Executive Secretary.

CHARLES F. MARTIN, M.D., *President*
Montreal, P. Q., Canada

JAMES H. MEANS, M.D., *Chairman*
Boston, Mass.

E. R. LOVELAND, *Executive Secretary*
133-135 South 36th Street, Philadelphia, Pa.